Canadian Workshop on Fusarium Head Blight/ Colloque Canadien Sur La Fusariose

Holiday Inn Crown Plaza
Winnipeg, Manitoba
November 28 - 30, 1999.

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Bill Legge
Brent McCallum
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Proceedings Compiled by Randy Clear, Grain Research Laboratory, Canadian Grain Commission, 1404-303 Main St., Winnipeg, Manitoba

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CANADIAN WORKSHOP ON FUSARIUM HEAD BLIGHT /
COLLOQUE CANADIEN SUR LA FUSARIOSE

Dear workshop participant and grain industry colleague:

On the following pages you will find summaries of the oral and poster presentations given at the Canadian Workshop on Fusarium Head Blight / Colloque Canadien sur la Fusariose (*CWFHB/CCF*), Winnipeg, Manitoba, Canada, November 28-30, 1999.

The aim of the workshop was to gather representatives from all areas of the Canadian grains industry affected by fusarium head blight. Further goals of the workshop were to provide the latest information on the disease; begin a dialogue to identify and prioritize areas of concern requiring study; and make recommendations and propose solutions to this devastating problem.

A workshop of the magnitude of the CWFHB/CCF required the support, assistance and participation of a large number of individuals and companies. We would first like to thank the various sponsors of the workshop for their generous financial support. We also would like to thank the members of the National and Local Organizing Committees who worked so hard to organize and conduct the event and ensure that it ran smoothly. Many of you, experts in your field, were approached and kindly agreed to make the time and effort to prepare and present the excellent and wide-ranging talks and posters. We are also grateful to the chairpersons of the various sessions, including the 'break-out' discussion groups, for their efforts in organizing and summarizing what took place. We wish to thank the Canadian Grain Commission for the satellite workshops offered on *Fusarium* identification and DON testing protocols which proved to be so popular. Last, but not least, we want to thank all 242 of you who attended *CWFHB/CCF*; your presence, participation, and complimentary remarks at its conclusion, were most gratifying to the National and Local Organizing Committees. Clearly, all of you were responsible for making the workshop a success!

As an outcome of the workshop, a recommendation was made (see page 107) that a Steering Committee be struck, composed of the members of the National Organizing Committee with the addition of other relevant representatives, to explore a coordinated and collaborative approach for obtaining supplementary funding to pursue the many research studies necessary to combat and vanquish this formidable foe. The motion to take this course of action was passed unanimously.

We would be glad for any additional comments or suggestions relating to CWFHB/CCF, and your opinions on the need and time-frame for a follow-up workshop, in future.

Yours cordially,

Malcolm Morrison Andy Tekauz
Co-chairs *CWFHB/CCF*

101-00059/

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Presentation Summaries

Type 2 Resistance & Other Thoughts on Fusarium

J. David Miller
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1. **Head blight is caused by several species**: *F. graminearum* and *F. culmorum* are closely related species that produce deoxynivalenol or nivalenol and zearalenone depending on geographic origin of the isolate (Miller et al. 1991). Resistance to *F. graminearum* provides resistance to *F. culmorum,* and, as long as toxigenic strains are used, there has been so sign of "race" differences between isolates of these fungi (Mesterhazy et al. 1999; Snijders 1994). *F. crookwellense* occasionally causes epidemics of head blight in wheat (Miller 1994). I know of no data to determine whether the horizontal resistance to the other species applies here. These fungi cause Fusarium head blight in small grains and Gibberella ear rot in maize. These diseases are associated with temperate grain-growing regions. Which species will dominate depends on temperature. *F. graminearum* is associated with cereals grown in warmer areas and *F. culmorum*, in cooler areas. *F. graminearum* was common in wheat from North America and China (Wang & Miller 1988a; Miller 1994). *F. culmorum* was the dominant species in cooler wheat growing areas such as Finland, France, Poland and The Netherlands (Miller 1994).

2. **Deoxynivalenol is more toxic and important than meets the eye**: There are only five agriculturally-important mycotoxins: deoxynivalenol, aflatoxin, fumonisin, ochratoxin and zealalenone. The discovery of deoxynivalenol as a widespread contaminant of wheat in the northeast of the USA and in eastern Canada in late 1979-81 focused scientific attention on toxins from *Fusarium* species. "Red mold poisoning" was reported in rural Japan throughout the 1950's which lead to investigations on the cause. Eventually, deoxynivalenol was discovered by Japanese researchers from grain that had made humans ill (Morooka et al. 1972). The same chemical was subsequently re-reported as "vomitoxin" by Ron Vesonder and colleagues studying problems in swine fed *F. graminearum* -contaminated corn in 1973 (Vesonder et al. 1973).

Deoxynivalenol is the most important mycotoxin on a world-wide basis in terms of human exposure (Miller 1995; IARC 1993). Except under dryland conditions, deoxynivalenol is found in wheat, the most important staple crop, world-wide. This toxin was not among the first discovered because of its relatively lower acute toxicity (Table 1). Much early research on trichothecenes was done by bioassaying fractions on the skins of rats and rabbits. Dermal sensitivity is similar to that of cultured cells (Ueno 1983) and deoxynivalenol is 47 and 70 times less toxic than DAS and T-2 toxin, respectively in BHK-1 cells (Rotter et al. 1993).

Large scale human toxicoses ascribed to deoxynivalenol have been reported in India and other parts of the world (Bhat et al. 1989; Miller 1990).

Table 1. Toxicities of some trichothecenes

toxin	mouse LD 50 (IP)		CD50 in BHK-1	
	mg/kg bw	ratio vs. T-2	ng/ml	ratio vs. T-2
T-2 toxin	5.2	1.0	1.6	1.0
DAS	23.0	4.4	2.4	1.5
nivalenol	4.1	0.8	84	53
deoxynivalenol	70.0	13.5	112	70
15-acetyl deoxynivalenol	14.0	2.5	896	560
3-acetyl deoxynivalenol	34.0	6.5	2347	1467

After Forsell et al. 1987; Rotter et al. 1993; Ueno 1983)

The fungi that produce deoxynivalenol, *F. graminearum* and *F. culmorum*, had been recognized to include isolates that were not typical of either. This species, *F. crookwellense*, produces nivalenol and related compounds, not deoxynivalenol (Lauren et al. 1987).

Trichothecenes are potent low molecular weight inhibitors of protein synthesis (Feinberg & MacLaughlin 1989). In addition, they cause physical damage to membranes resulting in cell lysis. Red blood cells are a compartment for trichothecene metabolism and these cells will lyse in the presence of excess circulating toxin. The amount of toxin required to lyse red blood cells varies according to animal species (Khachatourians 1990).

Although deoxynivalenol is less acutely toxic than T-2 toxin and DAS, the immunotoxic and neurotoxic properties of trichothecenes are of greater practical importance than the spectacular haemorrhages caused by the aforementioned toxins . Changes in immune system function in male mice occur at dietary concentrations often encountered by humans. As with other trichothecenes, high exposures increase susceptibility to facultative pathogens such as *Listeria*. Deoxynivalenol exposure produces prolonged elevations in serum IgA and mesangial IgA leading to hematuria (Pestka & Bondy 1994). Human IgA disregulation (Berger's disease) is common and the only agents so far demonstrated to reproduce this condition in experimental animals are trichothecenes (Pestka & Bondy 1994; Yan et al. 1998).

Feed refusal in swine is caused by the neurotoxic effect of deoxynivalenol. Experiments involving the dosing of the toxin by a continuous exposure osmotic pump (IP) resolved that the effects could not be due to taste or learned responses (Prelusky 1997). A single dose of 0.25 mg/kg (IV) changed neurotransmitter concentrations in the hypothalamus, frontal cortex, and cerebellum up to 8 d post dosing (Prelusky et al. 1992). A very low

dose (10 µg/k) IV resulted in changes in cerebral spinal fluid neurotransmitters. Based on acute human exposure-emesis data, humans are not less and are probably more sensitive to deoxynivalenol than swine (see Kuiper-Goodman 1994). Feed refusal also occurs in mice and in lifetime studies, this reduced the incidence of spontaneous liver tumours due to calory restriction (Iverson et al. 1995). Finally, the occurrence of deoxynivalenol in diets affects uptake of sugars and minerals (Hunder et al, 1991).

Deoxynivalenol is regulated by Health Canada and the United States Food & Drug Administration at similar levels in flour for human consumption. The FDA and the Canadian Food Inspection Agency have similar guidelines for the presence of deoxynivalenol in domestic animal feeds (Kuiper-Goodman 1994). One purpose of the animal feed guidelines is to prevent residues of known and unknown compounds in milk, meat and eggs.

3. **Epidemic conditions are associated with the planting of susceptible cultivars**: In Ontario, the more frequent appearance of Fusarium head blight occurred during a time when winter wheat cultivars became more susceptible. Frederick, the dominant cultivar in the early 1980's was much more resistant than the cultivars that followed; indeed it was intermediate between the susceptible and resistant materials that we tested with Dexter Samson in 1983-84 (Miller & Young 1986; Miller et al. 1984). Schroeder & Christensen (1963) wrote "The general practice of growing var. Marquis in the hard red spring region of the U.S. beginning about 1916, was accompanied by a pronounced increase in damage from scab." Data from Dr. Art Schaafsma's studies of the 1996 epidemic in Ontario (this meeting) and of our studies on tillage practice show that cultivar is the dominant variable. However, improper rotation practices are also an important variable (Miller et al. 1998).

4. **It is biologically plausible that the population of *F. graminearum* strains has changed**. It was proposed by Dr. Don Wicklow more than a decade ago that current agricultural practice should have the effect of increasing the prevalence of pathogenic or toxigenic strains. There is evidence for this hypthesis for *Aspergillus flavus* (Horn & Dorner 1999). Without access to well-preserved strains from 50 years ago, it is not possible to unambiguously resolve whether the prevalence of more pathogenic versus saprophytic strains of *F. graminearum* has increased. However, this is one explanation of Randy Clear's observations on the frequency of occurrence of this species on surface sterilized kernels.

4. ***Fusarium graminearum* is a necrotrophic pathogen**: Such pathogens invade by killing the host cells in advance. This was reported by the earliest investigators (see Schroeder & Christensen 1963 and references therein). These researchers and others reported no evidence of defensive host reactions.

The studies of Schroeder & Christensen done through the 1950s employed the cultivar Frontana, one that I also became interested in thirty years later. Frontana is a selection of Mentana (Italy) x Centenario (Uruguay) chosen for good yield after late planting around 1943 (Beckman, IX Int Genetics Meeting, Cornell, 1953). Schroeder & Christensen

(1963; done in 1953-1955) found that their accession of Frontana had resistance to initial infection (~ 10% of worst) and resistance to hyphal spread (~ 10% of worst). Wang et al. found about the same result (Wang & Miller 1988a). Schroeder and Christensen found no evidence of structural (histological) resistance factors leading to the conclusion that the difference between Frontana and the others was "physiological".

Wang YuZong, Dexter Samson, Chris Young and I inoculated various cultivars and measured symptoms, the amounts of deoxynivalenol and ergosterol present (Miller et al. 1984; Wang & Miller 1988b). In these tests cultivars with equivalent symptom expression or fungal biomass had up to 2-10 fold differences in the amount of deoxynivalenol present. This led us to the conclusion that there were factors that either prevented the synthesis of deoxynivalenol or degraded it or both (Miller et al. 1984). In fact, the very first measurements of deoxynivalenol under field conditions in 1983 in Ontario by Abe Teich, Peter Scott and Gordon Neish showed that the toxin declined (Scott et al. 1984) and thus the symptoms present in wheat did not strictly relate to the deoxynivalenol present. Although groups all over the world ultimately reported similar findings (see Mesterhazy et al. 1999), this issue was largely ignored by breeders.

Charles Snijders at the DLO in the Netherlands and I became interested in the role of deoxynivalenol as a virulence factor. Trichothecenes were recognized to be phytotoxic during the course of their initial discovery (Brian et al. 1961). It was not until our work on the phytotoxicity of deoxynivalenol was explored that it was realized that there was a strong difference in response between wheat cultivars resistant to Fusarium head blight. Coleoptile tissue of cultivars that were resistant to Fusarium head blight were 10 times more resistant to deoxynivalenol (and some other metabolites including 3 acetyl deoxynivalenol and dihydroxycalonectrin) than disease susceptible cultivars (Wang & Miller 1988b). This was shown to be due to the presence of a modified peptidyl transferase [at protein synthesis] (Miller & Ewen 1997) and to unknown functional changes in the membranes of more resistant types (Cossette & Miller 1995; Miller & Ewen 1997; Snijders & Kreching 1992). Earlier studies had shown that cultivars of wheat in the field appeared to be able to metabolize deoxynivalenol (Miller & Young 1985; Scott et al. 1984 among others) shown later to be the case *in vitro* in head blight-resistant cultivars (Miller & Arnison 1986). Strains that produce high concentrations of deoxynivalenol in the field were more virulent (Atanssov et al. 1994; Snijders 1994; Mesterhazy et al. 1999). This implied that one component of resistance to Fusarium head blight related to reducing the phytotoxic impact of deoxynivalenol. This has been examined from the other perspective i.e. by showing that strains of *F. graminearum* with the trichodiene synthetase removed have reduced virulence (Desjardins et al. 1996). Most crucially, deoxynivalenol results in massive electrolyte loss in plant cells upon exposure. This means that this necrotrophic pathogen produces a compound that results in cell lysis and thus the release of sugars, food for the fungus.

5. Type 2 resistance is mostly resistance to deoxynivalenol

Snijders and colleagues showed that deoxynivalenol appeared in wheat kernels in advance of fungal mycelia (Snijders & Perkowski 1990; Snijders & Kreching 1992):

infected spikes	susceptible		resistant	
	ERG	DON	ERG	DON
0	-	76ppm	16	-
7.5%	5	22	15	-
17.5	6	62	11	29
27.5	10	61	15	21
57.5	26	164	27	77

These findings led both Snijders and I to study electrolyte loss in the cultivars we worked on. As noted above, Frontana is substantially more resistant to the membrane damaging effects of deoxynivalenol than susceptible cultivars (Miller & Ewen 1997). Snijders tested many breeding lines and sources of resistant germplasm and found that leaf tissue from resistant lines from all geographic origins were resistant to 10^{-3} M deoxynivalenol. He found that correlation analysis between resistance to the phytotoxic effect of deoxynivalenol explained 75% of the variance of kernel ergosterol and deoxynivalenol contents (Snijders & Schepers 1993; 1994):

tissue	content	FHB	TKW reduction
chaff	ergosterol	0.80	0.86
	DON	0.82	0.91
kernel	ergosterol	0.62	0.70
	DON	0.50	0.60
FHB			0.90

As Schroeder & Christensen reported 40 years ago, resistance to Fusarium head blight appears mainly physiological in nature. Singh et al. (1995) suggested that there were 3-5 important genes for fusarium head blight resistance from Frontana germplasm. In the Frontana/Frederick, Frontana/Augusta crosses made by Dexter Samson, we found that trichothecene tolerance involved 3 genes.

6. Occupation exposures to dusts from Fusarium head blight may be more dangerous than we thought

From the beginnings of the Fusarium mycotoxin program at the Central Experimental Farm, Dr. Locksley Trenholm recognized the need to be careful handing grains and feeds contaminated with mycotoxins. By 1983, there were written CFAR guidelines on this issue. This perhaps was in part due to the knowledge that epidemiological studies of feed workers handling grains contaminated with aflatoxin had elevated risks for liver cancer. This was unambiguously shown only in 1993 (IARC 1993). Further, the US Army had supported considerable research on the impact of inhalation exposure to pure trichothecenes. Such exposures were found to be 20-50 times more potent than iv or ip exposures (Creasia et al. 1989). In the AAFC publication "Reducing mycotoxins in animal feeds" (1988), there are 3 pages of information on occupational issues.

In 1993, Labour Canada pondered requiring personal protection for workers handling *Fusarium*-contaminated grains. With Al Pighin and Francois De Mers of the then Labour Canada and Dr. Tina Kuiper-Goodman of Health Canada, we worked to provide data on trichothecene exposures in grain elevators. This provided a basis to undertake a risk assessment (De Mers 1994). We felt that the 1993-94 exposures did not pose a material risk for toxin-induced disease.

Since then, Finnish workers reported the concentrations of dusts, deoxynivalenol and spores associated with on-farm handing of grains. Deoxynivalenol contents were similar to those we had found in grain from the 1993 western crop (de Mers 1994; Lappalainen et al. 1996):

grain drying	milling	cattle feeding
10^7 spores/m^3	10^6 spores/m^3	10^6 spores/m^3
1 mg/m^3	2 mg/^{3}	1.4 mg/m^3

Our studies from the handling of the 1993 crop found 0.5 to 5.8 ppm DON + 1 ppm T2 + HT2 toxin in such airborne dusts. Canadian grain handlers were exposed to 2-6 times the airborne dust values associated with farming activities, at least in Finland.

Also since our analysis, Norwegian epidemiologists have been studying occupational disease in cereal farmers. In Norway, grains are more or less exclusively contaminated by *Fusarium* toxins essentially similar to the array known from Canadian grains. Perinatal health in woman farmers was at greater risk after harvest and after a poor crop. Occupational exposure to mycotoxins in grain was associated with miscarriage at an early stage of pregnancy (odds ratio 1.67-2.85; Kristensen et al. 1997). Submitted data from this group suggest that there are also non-respiratory health impacts on male farmers. Extensive exposure data are in the process of collection and further refinements of

possible association are anticipated in 2000 (Kristensen, personal communication).

This means that the theoretical risk of non-respiratory (i.e. toxins associated) diseases from long-term handling of *Fusarium* contaminated grain that we thought existed in 1994 are apparently low, but may be real.

7. The 1999 FAO/WHO/UNEP Tunis conference on mycotoxins

The report is:
(http://www.fao.org/WAICENT/FAOINFO/ECONOMIC/ESN/mycoto/mycoto.htm).
This was the third such meeting since 1977. Unlike the previous meetings, there was much more attention to the human health impacts of mycotoxins. Some 50 countries were formally represented and many resolutions were passed. Two working groups passed resolutions on the need for more attention to planting crops and genotypes susceptible to mycotoxin accumulation based on the impact on human health. This is, in my view, a warning that should be heeded to by agriculture. The conference identified the urgent need for a special meeting of the Joint Expert Committee on Food Additives and Contaminants on mycotoxins. It is anticipated that this will happen in 2001.

I thank Dilantha Fernando for suggesting that I be invited to speak at this important meeting.

Literature Cited

Atanassov, Z., C. Nakamura, N. Mori and C. Kaneda. 1994. Mycotoxin production and pathogenicity of *Fusarium* species and wheat resistance to Fusarium head blight. Can J Botany 72:161-167.

Bhat, R.V., S.R. Beedu, Y. Ramakrishna and K.L. Munshi. 1989. Outbreak of trichothecene mycotoxicosis associated with the consumption of mould-damaged wheat products in Kashmire Valley, India. Lancet, January 7, 1989:35-37.

Brian, P.W., A.W. Dawkins, J.F. Grove, H.G. Hemming, D. Lowe and G.L.F. Norris. 1961. Phytotoxic compounds produced by *Fusarium equiseti*. J Exp Botany 12:1-12.

Cossette, F. and J.D. Miller. 1995. Phytotoxic effect of deoxynivalenol and Gibberella ear rot resistance of corn. Natural Toxin. 3: 383-388.

Creasia, D.A. and R.J. Lambert. 1989. Acute respiratory tract toxicity of the trichothecene mycotoxin T-2 toxin. In: Beasley, V.R. (ed). Trichothecene mycotoxin mycotoxins: pathophysiologic effects, vol. 1. CRC Press, Boca Raton, FL. pp. 161-170.

De Mers, F. 1994. Preliminary assessment of the risk of exposure to trichothecenes produced by *Fusarium graminearum* in grain dust. Occupational Safety and Health

Branch, HRD, Ottawa.

Desjardins, A.E., R.H. Proctor, G. Bai, S.P. McCormicj, G. Shaner, G. Buechley and T.M. Hohn. 1996. Reduced virulence of trichothecene non-producing mutants of *Gibberella zeae* in wheat field tests. Molecular Plant-Microbe Interactions 9:775-781.

Feinberg, B, and C.S. MacLaughlin. 1989. Biochemical mechanism of action of trichothecene mycotoxins. In: Beasley, V.R. (ed) Trichothecene mycotoxin mycotoxins: pathophysiologic effects, vol. 1. Boca Raton, FL: CRC Press pp. 27-36.

Forsell, J.H., R. Jensen, J.H. Tai, M. Witt, W.S. Lin and J.J. Pestka. 1987. Comparison of acute toxicities of deoxynivalenol, (vomitoxin) and 15-acetyl deoxynivalenol in the B6C3F1 mouse. Food Chem Toxicology 25:155-162.

Horn, B.W. and J.W. Dorner 1999. Regional differences in production of aflatoxin B1 and cycolopiazonic acid by soil isolates of *Aspergillus flavus* along a transect within the United States. Applied Environmental Microbiology 65:1444-1449.

Hunder, G. K. Schumann, G. Strugala, J. Gropp, B. Fichtyl and W. Forth. 1991. Influence of subchronic exposure to low dietary deoxynivalenol, a trichothecene mycotoxin, on intestinal absorption of nutrients in mice. Food Chem Toxicol 29:809-814.

IARC 1993. Monograph 56: Some naturally-occurring substances: some food items and constituents, heterocyclic amines and mycotoxins. International Agency for Research on Cancer, Lyon, France.

Iverson F., C. Armstrong, E. Nera, J. Truelove, S. Fernie, P. Scott, R. Stapley, S. Hayward and S. Gunner. 1995. Chronic feeding study of deoxynivalenol in B6C3F1 male and female mice. Teratogenesis, Carcinogenesis and Mutagenesis 15:283-306.

Khachatourians, G.C. 1990. Metabolic effects of the trichothecene T-2 toxin. Can J Physiol Pharmacol 68:1004-1008.

Kuiper-Goodman, T. 1994. Prevention of human mycotoxicoses through risk assessment and risk management. In: J.D. Miller & H.L. Trenholm (eds), Mycotoxins in grain: compounds other than aflatoxin. Eagan Press, St. Paul, MN. pp. 439-470.

Kristensen, P., L.M. Irgens, A. Andersen, A.S. Bye, and L. Sundheim, L. 1997. Gestational age, birth weight and perinatal death among births to Norwegian farmers. American J Epidemiology 146:329-338.

Lappalainen, S., M. Nikulin, S. Berg, P. Parikki, E-L. Hintikka and A-L. Pasanen. 1996. *Fusarium* toxins and fungi associated with handling of grain on eight Finnish Farms. Atmospheric Environment 30:3059-3065.

Lauren, D.R., A. Ashley, B.A. Blackwell, R. Greenhalgh, J.D. Miller and G.A. Neish. 1987. Trichothecenes produced by *Fusarium crookwellense* DAOM 193611 in liquid culture. J. Agric. Food Chem. 35:884-889.

Mesterhazy, A., T. Bartok, C.G. Mirocha and R. Komoroczy. 1999. Nature of wheat resistance to Fusarium head blight and the role of deoxynivalenol for breeding. Plant Breeding 118:97-110.

Miller, J.D. 1990. Contamination of food by *Fusarium* toxins: studies from Austral-Asia. Proc. Japanese Association of Mycotoxicology 32: 17-24.

Miller, J.D. 1994. Epidemiology of *Fusarium graminearum* diseases of wheat and corn. In: J.D. Miller & H.L. Trenholm (eds), Mycotoxins in grain: compounds other than aflatoxin. Eagan Press, St. Paul, MN. pp. 19-36.

Miller, J.D. 1995. Fungi and mycotoxins in grain: implications for stored product research. J. Stored Product Research. 31: 1-6.

Miller, J.D., J.C. Young and D.R. Sampson. 1984. Deoxynivalenol and Fusarium head blight resistance in spring cereals. Phytopathol. Zeitschrift. 113: 359-367.

Miller, J.D. and J.C. Young 1985. Deoxynivalenol in an experimental *Fusarium graminearum* infection of wheat. Can J Plant Pathology 8:147-150.

Miller, J.D. and P.G. Arnison. 1986. Degradation of deoxynivalenol by suspension cultures of the Fusarium head blight resistant cultivar Frontana. Can. J. Plant Pathol. 8: 147-150.

Miller, J.D., R. Greenhalgh, Y.Z. Wang and M. Lu. 1991. Mycotoxin chemotypes of three *Fusarium* species. Mycologia 83: 121-130.

Miller, J.D. and M.A. Ewen 1997. Toxic effects of deoxynivalenol on ribosomes and tissues of the spring wheat cultivars Frontana and Casavant. Natural Toxins 5:234-237.

Miller, J.D., J. Culley, K. Fraser, S. Hubbard, F. Meloche, T. Ouellet, L. Seaman, K.A. Seifert, K. Turkington, and H. Voldeng. 1998. Effect of tillage practice on fusarium head blight of wheat. Can. J. Plant Pathol. 20:95-103.

Morooka, N., N. Uratsuji, T. Yoshizawa and H. Yamamoto. 1972. Studies on the toxic substances in barley infected with *Fusarium* species. J. Food Hygiene Soc Japan 13:368-375. [in Japanese].

Pestka, J.J. and G.S. Bondy. 1994. Mycotoxin-induced immune modulation. In: Dean, J.H., M.I. Luster, A.E. Munson and I. Kimber (eds) Immunotoxicology and Immunopharmacology. Raven Press, NY. pp. 163-182.

Prelusky, D.B. 1997. Effect of intraperitoneal infusium of deoxynivalenol on feed consumption and weight gain in the pig. Natural Toxins 5:121-125.

Prelusky, D.B., J.M. Yeung, B.K. Thompson and H.L. Trenholm. 1992. Effect of deoxynivalenol on neurotransmitters in discrete regions of swine brain. Arch Environ Contam Toxicol. 22:36-40.

Rotter, B.A., B.K. Thompson, S. Clarkin and T.C. Owen. 1995. Rapid colorimetric bioassay for screening of *Fusarium* mycotoxins. Natural Toxins 1:303-307.

Schroeder, H.W. and J.J. Christensen. 1963. Factors affecting resistance of wheat to scab caused by *Gibberella zeae*. Phytopathology 53:831-838.

Scott, P.M., K. Nelson, S.R. Khanhere, K.F. Karpinski, S. Hayward, G.A. Neish and A. Teich. 1984. Decline in deoxynivalenol (vomitoxin) concentrations in 1983 Ontario winter wheat before harvest. Appl. Environ. Microbiol. 884-886.

Singh R.P., H. Ma and S. Rajaram. 1995. Genetic resistance to scab in spring wheat cultivar Frontana. Plant Disease 79:238-240.

Snijders, C.H.A. 1994. Breeding for resistance to *Fusarium* in wheat and maize. In: J.D. Miller & H.L. Trenholm (eds), Mycotoxins in grain: compounds other than aflatoxin. Eagan Press, St. Paul, MN. pp. 37-58.

Snijders, C.H.A. and J. Perkowski 1990. Effects of head blight caused by *Fusarium culmorum* on toxin content and weight of wheat kernels. Phytopathology 80:566-570.

Snijders, C.H.A. and C.F. Krechting. 1992. Inhibition of deoxynivalenol translocation and fungal colonization in Fusarium head blight resistant wheat. Can. J. Botany 70:1570-1576.

Snijders C.H.A. and J. Schepers. 1993. The effect of deoxynivalenol of electrolyte leakage of wheat tissue with regard to Fusarium head blight resistance. Abstract, International Plant Pathology Congress, Montreal.

Snijders, C.H.A. and J. Schepers (1994). Cell membrane stability of wheat tissue in the presence of deoxynivalenol as a measure for Fusarium head blight resistance. DLO, Wageningen, The Netherlands.

Ueno, Y. 1983. Trichothecenes. Elsevier, Amsterdam.

Vesonder, R.F, A. Ceigler and A.H. Jensen. 1973. Isolation of the emetic principle from *Fusarium graminearum*-infected corn. Applied Microbiology 26:1008-1010.

Wang, Y.Z. and J.D. Miller. 1988a. Screening techniques and sources of resistance to Fusarium head blight. In: Wheat Production constraints in tropical environments. Edited by A.R. Klatt. CIMMYT, Mexico, D.F. p. 239-250.

Wang, Y-Z. and J.D. Miller. 1988b. Effects of *Fusarium graminearum* metabolites on wheat tissue in relation to Fusarium head blight resistance. J. Phytopathology. 122: 118-125.

Yan, D., H-R. Zhou, K.H. Brooks and J.J. Pestka. 1998. Role of macrophages in elevated IgA and IL1 production by Peyer's patch cultures following acute oral vomitoxin exposure. Toxicology and Applied Pharmacology 158:261-273.

Overview of the Fusarium Situation in Canada

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Fusarium graminearum and other *Fusarium* sp. cause head blight on wheat, barley and oats, and ear rot of corn. Since the early 1980's this disease has become one of the most important diseases of small grains in North America. Fusarium head blight (FHB) causes yield loss and reduces the grain quality. Floret failure and poor seed filling reduces yields. A disease that was once sporadic in eastern Canada has become a major recurrent problem in the Canadian prairies, especially in Manitoba. Since 1993. There have been several epidemics of FHB on wheat with losses to the cereal industry in Canada estimated at over $ 1 billion over the last seven years. The disease not only causes a yield loss by producing fusarium damaged kernels (FDK), but also has a profound effect on the feed, malting and brewing industries as it produces mycotoxins known as deoxynivalenol (DON). The pathogen has been spreading towards the west with noticeable losses reported from Saskatchewan, and the identification of *Fusarium graminearum* in fields of wheat and barley in several locations in Alberta. Since the early 1980's, when the first outbreak of any significance occurred in Canada, and Sutton (1982) wrote an excellent review of the situation, we have come a long way in understanding the effects of mycotoxins (Miller, 1994), the epidemiology (Paulitz, 1996) and the nature of resistance (Buerstmayr et al., 1999) in wheat to FHB. For example, in the knowledge of epidemiology, the importance of ascospores in the disease cycle of the FHB pathogen has been investigated by several researchers (Paulitz 1996, Fernando et al., 2000). The release of ascospores with peak numbers trapped in the evening between 1600 h and midnight was reported by Paulitz in 1996. Location, year, or size of plot did not affect this diurnal pattern of spore release. Paulitz, speculated that the increase in RH in the evening following drying during the day could increase the turgor pressure of asci. Fernando et al. (2000) in their work trapped both ascospores and macroconidia. Ascospores, but macroconidia, showed a daily periodicity. *Gibberella zeae* ascospores were recovered mostly at night and showed four main release events during the 20-day sampling period which was concurrent with anthesis. The spread of disease via ascospores in the prevailing wind direction has been suggested over spread of disease through macroconidia (Fernando et al., 1997). They studied the disease foci from an inoculum source. Comparison of conidial- and ascospore-derived disease gradients indicated a lack of secondary infection, confirming that Fusarium head blight is primarily a monocyclic disease. They concluded that ascospores to be responsible for primary infection and macrocondia splashed through rain, probably be important in the infection of secondary tillers. We have also made vast strides in understanding the genetics of resistance in wheat, using molecular markers (Procunier 1999. at this conference, see text).

The emergence of relatively new cultural practices such as no-till farming in the west may be contributing to the FHB problem. *Fusarium graminearum* survives well in crop

residue and with the right environmental conditions such as high humidity and rain fall during anthesis, the susceptible wheat varieties that are grown (no known resistant variety available) may be colonized and infected by the pathogen. Rotations away from corn, wheat and barley may help reduce disease (Gilbert and Tekauz, 1999). Another important factor to consider is the genetic diversity among the *Fusarium graminearum* isolates. The question remains whether this diversity has any significance in management of the pathogen.

The FHB disease was not a problem in barley until 1993 (Tekauz et al. 1999). However, there has been a gradual increase of the incidence and severity of the disease specially in Manitoba and by 1998 was as bad as in wheat (McCallum et al. 1999). Tekauz et al. 1999 in their review of FHB in barley argues that this may have resulted from a combination of factors such as, a fundamental shift in the pathogen population, new cultivars grown in the region, and environmental conditions promoting disease. The presence of fusarium species such as *F. poae, F. sporotrichioides*, and *F. avenaceum* have reduced with an increase of *F. graminearum* over the last few years. Also in the last five years, production of 6-rowed barley has increased in Manitoba, and these generally are more susceptible to FHB (Anon. 1998). The occurrence of FHB in barley has created new problems to the industry. The feed and malting industry has very low DON tolerance levels. The need for a fast, and accurate DON testing method would help the industry immensely.

The spread of disease to new areas is of great concern. There is compelling evidence that *F. graminearum* has recently been moving westward from southeastern Manitoba, replacing less pathogenic species as the principal FHB pathogen (Clear and Patrick, 1999). During 1998, in Manitoba, estimated losses in yield alone averaged 10% in wheat and 5.5% in barley. Again, there were localized outbreaks with variability from field to field. FHB occurred mainly in early seeded spring cereal crops and later seeded crops largely escaped infection. 1998 saw a dramatic rise in the incidence and severity of FHB in SE Saskatchewan, and for the first time FHB was of economic importance to any prairie region outside of Manitoba. Though *F. graminearum* has been isolated from several areas in Alberta, it has not been a significant threat to the wheat and barley crops. The movement of grain from Manitoba to Alberta may increase the chances of spread of pathogen to new areas. The significance of Fusarium that is seed-borne, and the fate of fusarium damaged kernels (FDK) coming out of the combine at harvest need to be understood to combat disease spread.

Though FHB is not easy to control, cultural, chemical, biological and genetic control have been used to reduce severity and spread. Cultural methods may reduce inoculum in crop residue, however if unusually wet summer weather that can lead to humid conditions during flowering and early stages of development, and inadequate resistance in wheat and barley cultivars together may trigger development of FHB to epidemic proportions. Chemical control has not been effective until Folicur 432F (tebuconazole) was introduced through emergency registration in the prairies in 1999. Several farmers have indicated that they were satisfied with the performance of the Folicur compound in several locations in Manitoba (personal communication). Research on biological control of FHB

is at its infancy. Though it may have some potential in reducing residue inoculum levels, control of ascospore infection at anthesis may be difficult. Given the right conditions (i.e. environment, variety), the pathogen requires minimum inoculum levels to cause significant damage. Genetic resistance if available would be the best for managing FHB. Resistance to FHB in wheat is quantitative and controlled by 2-5 genes. Extensive use of a few sources of resistance such as Sumai 3 and its Ning derivatives, and Frontana in wheat breeding programs throughout the world raises the question of selection pressure that it may have on the resistance genes. Several labs are now attempting to introduce additional resistance genes to the gene pool from wild wheat relatives. In barley we know very little of the make up of resistance. However, there is some tolerance to FHB available in cultivars AC-Oxbow, AC-Metcalfe, CDC-Stratus, CDC-Kendal and the hulless 6-row variety CDC-Silky. Several putatively resistant genotypes are also available from different parts of the world (i.e. Chevron from USA, Zhedar 1 from China and Seijo II from Japan). These genotypes could be used in crosses with our varieties to increase resistance to FHB in barley.

Ear rot of corn caused by *F. graminearum, F. moniliforme* and *F. subglutinans* is a problem in corn growing areas such as southern Ontario and in the corn belt states in the USA. The corn breeding program at the Eastern Cereal and Oilseed Research Centre in Ottawa have used an inbred line CO272 which has resistance to fusarium as the donor parent (Reid and Hamilton, 1999) . CO272 conforms to silk resistance with a thicker wax coating and a single dominant gene for resistance. Several inbreds have been released with resistance to fusarium ear rot (i.e. CO387, CO388 and CO389).

The long term impact of the pathogen is not well understood, but loss of yield and quality make control of this disease crucial to cereal producers. We must continue to monitor the spread of disease and make producers aware of management practices to reduce the impact of disease while breeding programs strive for better resistant varieties. We have come a long way in research on many aspects of the fusarium head blight problem, but still some areas remain in the research priority list. They are, breeding for resistance using marker assisted selection and wide crosses, and a better understanding of the epidemiology, seed infection and manure management. Also the need for a quick and reliable DON testing method still exists. Research funding, and a coordinated national research effort is required to tackle this FHB problem before it devastates new areas.

Literature Cited:

Anonymous. 1998. Manitoba Agriculture Variety Recommendations: In seed Manitoba 1999- Variety Recommendations and Growers Directory. The Manitoba Co-operator, Winnipeg, MB.

Buerstmayr, H., Lemmans, M., Fedak, G., and Ruckenbauer, P. 1999. Back-cross reciprocal analyisis of Fusarium head blight resistance in wheat (*Triticum aestivum* L.). *Theor. Appl. Genet.* 98:76-85.

Clear, R.M., and Patrick, S.K., 1999. The Fusarium head blight pathogens recovered from cereal seed in western Canada, 1993 to 1998. *Can. J. Plant Path.* (in press).

Fernando, W.G.D., Miller, J.D. Seaman, W.L., Seifert, K., and Paulitz, T.C. 2000. Daily and seasonal dynamics of airborne spores of *Fusarium graminearum* and other *Fusarium* species sampled over wheat plots. *Can. J. Bot.* (in press).

Fernando, W.G.D., Paulitz, T.C., Seaman, W.L., Dutilleul, P., and Miller, J.D. 1997. Head blight gradients caused by *Gibberella zeae* from area sources of inoculum in wheat field plots. *Phytopathology* 87:414-421.

Gilbert, J. and Tekauz, A. 1999. Recent developments in research on fusarium head blight of wheat in Canada. *Can. J. Plant Path.* (in press).

McCallum, B. Tekauz, A., Gilbert, J., Mueller, E., Kaethler, R., Stulzer, M., and Kromer, U. 1999. Fusarium head blight of barley in Manitoba in 1998. *Can. Plant Dis. Surv.* (electronic) 79:75-76.

Miller, J. D. 1994. Epidemiology of Fusarium ear rot diseases of cereals. Pages 19-36 in: Mycotoxins in Grain: Compounds other than Aflatoxins. J.D. miller and H.L Trenholm, eds. American Association of Cereal Chemists, St. Paul, MN.

Paulitz, T.C. 1996. Diurnal release of ascospores by *Gibberella zeae* in inoculated wheat plots. *Plant Dis.* 80:674-678.

Paulitz, T.C., Dutilleul, P., Yamasaki, S.H., Fernando, W.G.D.,, and Seaman, W.L. 1999. A generalized two-dimensional Gaussian model of disease foci of head blight of wheat caused by *Gibberella zeae*. *Phytopathology* 89:74-83.

Reid, L.M., and Hamilton, R.I. 1999. Advances in research: the development of improved ear rot resistance in corn. ECORC - http://res.agr.ca/ecorc/program/research/advances/res-02.num

Tekauz, A., McCallum, B., and Gilbert, J. 1999. Fusarium head blight of barley in western Canada: a review. *Can. J. Plant Path.* (in press).

A Summary of the USA Situation on FHB

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Here at the end of the 1990's, Fusarium Head Blight (FHB) has become a crop disease of great visibility in U. S. agriculture. All groups dealing with small grains, from farmers and grain handlers to the corporate giants in the milling and brewing industries, are concerned about some aspect of FHB. In the agriculture research community FHB has attracted attention not only among plant pathologists and plant breeders, but by cereal scientists and others concerned with food safety (Stack 1999). Not since the Southern Corn Leaf Blight outbreak in the early 1970's or wheat Stem Rust in the 1950's, has a particular crop disease gotten so much attention. Perhaps only Dutch Elm Disease has a higher public name recognition, although not as a threat to peoples' livelihood and businesses.

With 20-20 hindsight we can see a widespread trend of increasing FHB in the USA through the past two decades; a trend which began with outbreaks in Minnesota, Kansas, Nebraska, Indiana, North Dakota, and elsewhere in the early and mid 1980's (McMullen et al 1997). After the severe epidemics of 1993-1997 in the northern spring grain region and that of 1996 in the soft winter wheat region in the great lakes states, as well as occurrences in other states, FHB became a national concern. A map of FHB in the USA shown by McMullen et al (1997) reveals that nearly every wheat producing state except those in the arid west has seen FHB during the past decade.

What had begun in 1993 as a regional forum on FHB among scientists, farmers, and industry in the spring grain states became, in 1997, the first National FHB forum. The proceedings of the succeeding 1998 and 1999 National FHB forums are available on-line at [www.scabusa.org]. In preparing for this presentation, I reviewed the papers and discussions in that first regional forum of 1993. I was struck by two things: first, how much our knowledge of FHB has progressed in the past six years; second, how much the topics and issues for debate on the program for this conference parallel those at that one (Wilcoxson 1993).

In the wake of the several local outbreaks, some states had appropriated or redirected modest sums to FHB research. Minnesota, following the 1993 FHB epidemic, made a multimillion dollar resource available to scientists to address the problem. By 1997, a group of leaders from the grain industry, farming, and research communities in several states had joined together to stimulate a national effort on FHB. Recognizing that dollars, not just discussion were vital to a solution, this group sought new funding for research. Through their efforts the US Congress appropriated funds for FHB, which in 1998-99 totaled $3.1 million for several different research areas, including crop breeding, plant disease epidemiology, biotechnology, chemical and biological control, and food safety.

Many areas of research have been stimulated by the increased funding, too many to completely review here. One already showing promising results is improved chemical control, using additional products, timing, and improved application technology. The search for sources of resistance has greatly expanded both in numbers of candidates evaluated and in the kinds of material, from collections of wheat and barley accessions to wild relatives, and to unrelated wild grasses. It already appears that there are many diverse sources, an outcome promising for stability of resistance. Efforts to obtain molecular markers for FHB resistance genes have been expanded and the possibility of forecasting FHB is being explored.

It has been said that even the darkest cloud has a silver lining. Certainly, FHB has been a dark cloud for many farmers and the grain industry. The very widespread nature of the problem, however, brings an opportunity which occurs with few plant diseases. Instead of begging for support for a research on subject only a few recognize as important, the general public recognition that there is a problem means resources will be made available to address it. This has happened in the US at the present.

References:

McMullen, M.P., Jones, R., and Gallenberg, D. 1997b. Scab of wheat and barley: A re-emerging disease of devastating impact. Plant Dis. 81:1340-1348.

Stack, R. W. 1999. Return of an old problem: Fusarium Head Blight of Small Grains. APSnet feature article. (www.scisoc.org/feature/FHB)

Wilcoxson, R.D. 1993. Historical overview of scab research. p.1-5 IN: Proc. (1st) Regional Scab Forum, Moorhead, MN. Publ. Minn. Wheat Res. & Prom. Council, Red Lake Falls, MN 96p.

Proc. National FHB Forum 1998, 1999. at: www.scabusa.org.

Wheat Scab in China: Breeding, Research, and Development

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Scab (*Gibberella zeae*) is one of the most severe diseases in China (second only to stripe rust), infecting approximately one quarter of the total wheat production area (or six million hectares). In short, China has the largest scab-infected area in the world. Wheat scab is most prevalent in Middle-Lower Yangtze plains and in Northeast China. Recently, wheat scab has spread to the northern part of Huai River and to Northwest China. Wheat scab occurs annually in the Middle-Lower Yangtze plains. There the average yield loss is 5-10%. In epidemic years, yield loss can be higher than 20% and the quality is so poor that the grains can no longer be used for human and livestock consumption (Jin 1983).

Significant progress has been made in breeding for scab resistance in China with many new varieties showing increased levels of resistance. Varietal resistance, however, is still required to be supplemented by application of such fungicides as carbendazim and tebuconazole, to achieve an effective control for scab. In fact, chemical control remains the principal method for controlling scab in China. Under the government's regulation, wheat crops must be sprayed with fungicides once, or twice at the flowering stage. Use of fungicides not only increases the production costs, it also contaminates the grains with residues. Greater resistance than those presently available is needed to help reduce the use of fungicides in controlling wheat scab.

China started to conduct research on wheat scab as early as in 1936 after a severe epidemic of scab in Jiangsu and Anhui. Early work involved collection and evaluation of resistant germplasm as well as collection and isolation of *Fusarium* species. Later in the1950s, Chinese scientists started to study the genetics of scab resistance and the virulence of *Fusarium* species, to breed for resistant varieties, and to develop the chemical control methods.

I. Collection and Evaluation Scab-Resistant Germplasms

Since the 1940s, Chinese scientists have collected and evaluated 34,571 wheat accessions for scab resistance. Of them, 23,434 accessions were local landraces/varieties and 9,184 accessions were introduced from other countries (Lu 2000). In 1974, the China Scab Research Group tested 17,621 accessions for scab resistance. Based on the test results, the Group classified these accessions into five classes: 32 resistant, 229 moderately resistant, 123 moderately resistant to moderately susceptible, 1,104 moderately susceptible, and 16,133 susceptible (China Scab Research Group 1984). The 32 resistant accessions included landraces such as Wangshuibai, Wenzhou hongheshan, Fanshan xiaomai, and Pinghu jianzimai as well as improved varieties such as Sumai 3, Ning 7840, Fan 60096, and Jingzhou 1. The resistance level of these varieties, particularly Wangshuibai and Sumai 3 are strong and stable over environments. When Wangshuibai was tested at nine sites, it was resistant to scab in 41 site-years and was

moderately resistant in 2 site-years. Likewise, Sumai 3 was resistant in 92 site-years, and moderately resistant in 5 site-years when it was tested at 11 sites. Both Wangshuibai and Sumai 3 are now widely used by many wheat breeding programs as sources of scab resistance in both inside and outside of China.

II. Genetic Analysis of Major Sources of Resistance

1. Number of genes . Since the 1980s, genetic control of scab resistance has been a subject of many studies. Many workers have studied the inheritance of scab resistance in Sumai 3, Wangshuibai, Ning 7840, Yangang fangzhu, Wenzhou hongheshan, Fanshan xiaomai, Fan 60096, and Frontana (Liao and Yu 1985, Bai and Xiao 1989, Wang et al. 1991, Singh et al. 1995, Bam 1996, Van Ginkel et al. 1996). It is generally agreed that scab resistance is controlled by multiple genes (Table 1). Results varied from study to study because of differences in testing methods and testing environments.

Table 1. Number of genes controlling scab resistance in some wheat varieties

Variety	No. of genes	References
Sumai 3	2-4	Zhou (1987), Bai et al. (1990), Wang et al. (1991), Lin and Yang (1992), Bam (1996)
Wangshuibai	2-6	Bai et al. (1990), Liao and Yu (1985), Lin and Yang (1992)
Ning 7840	2-4	Zhou et al. (1987), Bai and Xiao (1989), Wang et al. (1991), Van Ginkel et al. (1996)
Yangang fangzhu	3	Bai et al. (1990)
Wenzhou hongheshan	2	Bai et al. (1990)
Fanshan xiaomai	3-4	Wang et al.(1991)
Fan 60096	3-4	Wang et al. (1991)
Frontana	2-3	Singh et al. (1995), Van Ginkel et al. (1996)

2. Chromosomal locations. Several workers (Yu 1982 & 1991, Liao and Yu 1985, Li and Yu 1988) have used monosomic lines from Chinese Spring, and Yao (1997) used substitution lines from a Chinese Spring/Sumai 3 cross to determine the chromosomal locations for resistance genes in Sumai 3, Wangshuibai, Wenzhou hongheshang, and Yangang fangzhu. Table 2 shows that scab resistance genes are located on several chromosomes. The genetics of scab resistance is very complex as different varieties carry resistance genes on different chromosomes. As of now, there is no consensus on the chromosomal locations of the resistance genes of Sumai 3 because these studies involved different testing methods and testing environments. On the other hand, Yu (1982), Yao (1997), and Nicholson (1999, personal communication) all found that Chromosome 3D of Sumai 3 carried a gene or genes controlling scab susceptibility.

Table 2. Chromosomal locations of major sources of scab resistance

Variety	Monosomic lines analysis	Substitution lines analysis
Sumai 3	1B** 2A** 5A** 6B** 7D**	2B* 3B* 6B* 7A**
Wangshuibai	4A*** 4D* 5A* 7A* 7B*	
Wenzhou hongheshang	3D** 5B** 6B**7D**	
Yangang fangzhu	3A** 4D**	

*, **, *** Monosomic/substitution lines had fewer infected spikelets than Chinese Spring at the 0.05, 0.01, and 0.001, respectively.

III. Genetic Improvement for Scab Resistance

Genetic improvement for scab resistance in wheat was a major emphasis in the early fifties. Wheat breeders have successfully isolated pure lines from commercial varieties. For example, Wennian 2 and Wangmai 15 were isolated from Nandai 2419 (Mentana), and Wumai 1 and Yangmai 1 from Funo. In 1961, Hubei Academy of Agricultural Sciences developed Emai 6 from Nandai 2419 by ^{60}Co radiation. Since the 1960s, the Institute of Food Crops (Jiangsu Academy of Agricultural Sciences), Taihu Institute of Agricultural Sciences, and Yangzhou Institute of Agricultural Sciences have carried out extensive crossing programs and each developed a series of new varieties including Sumai 3. Jingzhou Institute of Agricultural Sciences in Hubei developed two scab-resistant varieties (Jingzhou 1 and Jingzhou 4) from a Nandai 2419/*Secale cereale* cross and Jingzhou 66 from a Funo/*Triticum durum* //Nandai 2419/ *S. cereale* cross. Many of these high-yielding, moderately scab-resistant varieties have been widely grown in the Middle-Lower Yangtze plains (Jin 1983).

IV. Biotechnological Breeding

As biotechnologys, particularly in plant cell engineering, advance and mature, more and more of these biotechnologys will be integrated into conventional breeding methods to increase the breeding efficiency.

1. Cell culture for induction of somaclonal variation. Plant somatic tissues under explant conditions will de-differentiate and re-differentiate, and may give rise to an extensive amount of genetic variation (McCoy et al. 1982, Larkin et al. 1984). Empirical results from a multiple-years study showed that somaclonal lines derived from calli from young inflorescence/immature embryo culture were different for many traits including plant height, heading date, fertility, spike and kernel characteristics, and disease resistance. A 1989 study indicated that the overall frequency of somaclonal variation ranged from 50.2 to 94.9% (Table 3). The frequency of somaclonal variation for powdery mildew resistance and scab resistance was approximately 6.0%. Despite that the changes in plant traits could be positive and negative, the changes for increased level of resistance, however, can be transmitted from generation to generation.

Table 3. Frequencies (%) of somaclonal variants in R2

Variety	Height	Heading	Fertility	Spike	Disease resistance	Kernel	Awn	Total
Alondra	27	21	12	13	5	7	4	91
Veer	28	23	14	9	6	8	6	95
Yangmai	22	14	9	9	10	4	5	74
Ningmai 3	18	18	8	9	9	6	4	72
Sumai 3	13	9	6	9	3	5	4	50
Mean	26	17	10	10	7	6	5	76

Many high-yielding, scab-resistance lines were regenerated from susceptible varieties during the period from 1986 to 1998. For example, Shengkang 1 (895004) and 894013 were callus-derived lines from the susceptible variety Ningmai 3. The former has been approved for commercial production and is being grown on 300,000 hectares across several provinces: Jiangsu, Hubei, Anhui, Zhejiang, and Shanghai (Lu et al. 1998). Another somaclonal line 894037 was regenerated from the susceptible variety Yangmai 3. Both 894013 and 894037 yielded higher than Sumai 3 and their scab resistance was as strong as Sumai 3. Both lines are now used by wheat breeders as parents in their crossing programs.

2. In vitro selection for tolerance to DON. Several workers have used toxins or crude toxins as the selection agent for mutant cells and successfully isolated disease-resistant variants in tobacco, sugarcane, maize, sugar beets and other crops (Hu et al. 1988). Zhang et al. (1991) obtained scab-resistant variants from susceptible parents through in vitro selection on a selection medium containing crude toxin extracted from *F. graminearum*. Zhou et al. (1993) found that the optimum concentration of toxin for in vitro selection was 0.6-0.8 X 10^{-4} mol/L. Using these concentrations, Lu et al. (1998) carried out in vitro selection for tolerance to DON on calli derived from young inflorescence and immature embryos. They obtained more than 10 moderately resistant variants from the susceptible variety Alondra. Among these variants, T9108 and 943115 had only 11.8 and 13.4% infected spikelets, respectively, which were lower than Alondra's rate (71.8%).

3. Embryo rescue to facilitate gene transfer. Embryo culture is an effective mean to facilitate gene transfer from wild species or another genera to wheat varieties. Early research indicated that wild rye (*Elymus gigantens*), *Elytrigia elongata,* rye (*S. cereale*), wheat grass (*Roegneria ciliaris* and *R. kamoji*), *Haynaldia villosa,* goat grass (*Aegilops squarrosa*) carry gene(s) resistant to initial infection (Type I resistance) and to spread of the pathogen (Type II resistance) (Mujeeb-Kazi and Rodriguez 1981, Weng and Liu 1989, Wang 1991, Wan 1997). The two common species in the Middle-Lower Yangtze plains, *R. kamoji* and *R. ciliaris*, in particular, shows strong resistance to scab. Since 1991, Lu et al. (1998) have initiated experiments with an attempt to transfer scab-resistance gene(s) from *R. kamoji* to Chinese Spring. The procedure involved spraying 0.1% 2,4-D at 24-48 hours after pollination and dissecting the immature embryos at 10-12 days after pollination. With this procedure, the frequencies of embryo formation and seedling production were approximately 5% and 20%, respectively. The fertility rate became stable after six backcrosses to Chinese Spring. Field evaluation showed that four of the backcross progenies were moderately resistant to scab. One of which, 983222 had a very

strong resistance with lower than 10% infected spikelets.

4. Induction and selection for scab-resistant, anther-derived plants. As mentioned earlier, Sumai 3 and Wangshuibai are widely used by wheat breeders as sources of scab resistance. These two varieties, however, are tall and produce low yield. Therefore, it is difficult to develop high-yielding, scab resistant lines from single crosses. Very often, wheat breeders adopt a complex crossing scheme involving multiple parents (Zhou et al. 1987). Anther culture can be used to obtain doubled-haploid (DH) lines homozygous for all scab-resistance loci. It can be used to shorten the time required for developing a new variety by 3-4 generations. Figure 1 illustrates the efficiency of the anther culture method for wheat breeding. The high-yielding, moderately scab-resistant variety Yangmai 5 was crossed with the low-yielding, scab-resistant variety Sumai 3 in 1997. The F1 hybrids were crossed with the high-yielding variety Yangmai 158 in Winter 1994. Anther culture was conducted in the following Spring. In 1997, a short, scab-resistant, and high-yielding line (962426) was identified from the DH lines, and in 1998 it was entered in the province-wide trial.

Yangmai 5 X Sumai 3	1994 Spring (field)
↓	
F1 X Yangmai 158	1994 Winter (greenhouse)
↓	
F1	1995 Spring (Anther culture)
↓	
F1H3	1997 Autumn
↓	
Ning 962426	1998 Autumn

Fig. 1 Breeding procedures of the anther-culture-derived line 962426.

In addition to time saving, anther culture can be used to pyramid scab-resistance genes from various background into a single variety. Wangshuibai was derived from a local variety in Piaoyin (Jiangsu), Sumai 3 from a cross between Funo and a Taiwan wheat variety, and 894037 from callus culture of Ningmai 3. To-date, anther-derived lines with strong and stable resistance to scab have been selected from crosses involving these three varieties. These lines yielded higher than Sumai 3 and Wangshuibai. DH lines with stronger resistance than Sumai 3 and Wangshuibai, however, are yet to be identified.

5. Alien gene transfer. Since the successful production of the first transgenic wheat in 1992, wheat transformation research has greatly expanded, many transformation techniques have been developed, and many target genes have been made available. However, gene transfer of scab resistance is just at its infancy. Genetic control for scab resistance is quite complex, and thus scab-resistance gene(s) are yet to be cloned. Currently, most of the gene transfer work involves self-defence genes and anti-fungal protein genes, such as Chitinase, β-1,3-glucanase, and *Gastrodia* Anti-Fungal Protein. Recently, we have attempted to transfer target genes by particle bombardment,

Agrobacterium, and pollen tube pathway. Although the frequency of transgenic plants was low, Zhou et al. (1999) were able to obtain some transgenic wheat plants by particle bombardment and Zheng et al (1994) by the pollen tube pathway method.

V. Molecular Marker Analysis for Scab Resistance

Fusarium infection and spread are dependent upon temperature and humidity. The degree of infection of one variety could be different under different inoculation conditions. Therefore, visual selection for scab resistance is difficult. Marker-assisted selection offers new opportunities to improve the selection efficiency for scab resistance

1. Construction of experimental populations. Since 1992, we have produced recombinant inbred lines from the six resistant/susceptible crosses: Sumai 3/Alondra, Sumai 3/Annong 8455, Wangshuibai/Alondra, Wangshuibai/Annong 8455, 894037/Alondra, and 894037/Annong 8455. We have also produced DH lines from the following two crosses: Sumai 3/Alondra and 894037/Alondra by the anther culture method. Yao et al. (1997) developed 21 single-chromosome substitution lines from a Chinese Spring/Sumai 3 cross. These experimental populations have laid the foundation for molecular marker analysis.

2. RFLP analysis. Since 1998, we at the Institute of Genetics and Physiology have examined the RFLP polymorphisms in Sumai 3, Wangshuibai, 894037, and Alondra. As of September 1999, we screened 516 probes and found that 99 of the 516 probes showed polymorphisms. The frequency of RFLP polymorphisms was 15% between Wangshuibai and Alondra, 12% between Sumai 3/Alondra, and 14% between 894037/Alondra. When plant DNA was digested by the restriction enzymes *Eco*RI, *Hin*dIII or BamIII and hybridized with KSUF11, the autoradiogram showed a common band for all three scab-resistant varieties (Wangshuibai, Sumai 3, and 894037) and no band for the susceptible variety Alondra. Whether or not this RFLP band is associated with scab resistance remains to be shown.

3. RAPD analysis. Shen et al. (1996) has studied RAPD polymorphisms on scab-resistant somaclonal line 894037 and its parent Yangmai 3. A total of 376 primers were screened, and seven of them showed polymorphisms (OPQ13, OPR08, OPR10, OPS10, OPT04, OPT11 and OPT16). These seven primers later were used to study RAPD polymorphisms between scab-resistant varieties and scab-susceptible varieties. Three RAPD bands (OPR08-1174, OPR10-1584, and OPR10-1683) appeared in scab-resistant varieties and none of these bands appeared in scab-susceptible varieties. Therefore, it is possible that these RAPD bands are associated with scab resistance. RAPD analysis for the above experimental populations is underway. At the same time, we also screened 420 primers on Sumai 3, Chinese Spring, and a Sumai 3/Chinese Spring substitution line with Chromosome 7A from Sumai 3 for RAPD polymorphisms. Two primers (OPF02 and OPH19) expressed polymorphisms. Linkage analysis will be carried out.

Looking back to the 50-year history of scab-resistance breeding, we noticed that screening and utilization of scab resistance genes, genetic studies on scab resistance, and breeding for scab resistance have gradually developed. Significant progress has been made in several areas: from collection of resistance germplasms to alien gene transfer, from pure-line selection to biotechnological breeding, and from biometrical genetics to gene mapping. As molecular biology and gene technology advance and are increasingly applied to scab resistance research, we should be able to increase our level of understanding of scab resistance and to achieve better results from breeding for scab resistance.

VI. Acknowledgements

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VII. References

Bai, G. H. and Q. P. Xiao. 1989. Acta Agric. Shanghai 5(4): 17-23.

Bai, G. H. et al. 1990. Pages 171-177. *In* L. H. Zhu (editor). Advances in genetical research on disease resistance in major crops. Jiangsu Science and Technology Press, Nanjing.

Bam, T. 1996. Pages 13-17. *In* Global status and future prospects. CIMMYT, Mexico. .

China Scab Research Group. 1984. Crop Germplasm Resources (4): 2-7.

Hu, H. et al. 1988. Plant somatic cell genetics and crop improvement. Beijing Agric. Univ. Press, Beijing. 268-284.

Jin, S. B. (editor-in-chief). 1983. Chinese wheat cultivars and their pedigrees. Agricultural Press, Beijing.

Larkin, P. J. et al. 1984. Theor. Applied Genet. 67: 443-455.

Li, Y. F. and Y. J. Yu. 1988. J. Huazhong Agric. Univ. 4(2):327-331.

Liao, Y. C. and Y. J. Yu. 1985. J. Huazhong Agric. Univ. 4(2): 6-14.

Lin, Y. B, and Z. P. Yang.1992. Acta Agric. Shanghai 8(1): 31-36.

Lu, W. Z. et al. 1998. Jiangsu J. Agric. Sci. 14 (1): 9-14.

Lu, W. Z. 2000. Wheat scab research. China Science and Technology Press, Beijing (in press).

McCoy, T. J. et al. 1982. Can. J. Genet. Cytol. 24: 37-50.

Mujeeb-Kazi, A. and R. Rodriguez. 1981. J. Heredity 72: 253-256.

Shen, X. R. et al. 1996. Jiangsu J. Agric. Sci. 12(1): 7-10.

Singh, R. P. et al. 1995, Plant Dis. 79: 238-240.

Van Ginkel, M. et al. 1996. Plant Dis. 80: 863-867.

Wan, Y. F. 1997. Acta Phytopathologica Sinica 27(2): 107-111.

Wang, Y. N. 1991. J. Nanjing Agric. Univ. 14(2): 1-5.

Wang, Y. P. et al. 1991. Jilin Agric. Sci. (1): 21-28.

Weng, Y. Q. and D. J. Liu. 1989. Scientia Agricultura Sinica 22(5): 1-7.

Yao, J. B. et al.1997. Acta Agronomica Sinica 23: 450-453.

Yu, Y. J. 1982. Cereal Res. Comm. 10: 185-189.

Yu, Y. J. 1991. Acta Agronmica Sinica 17: 248-254.
Zhang, Y. et al. 1991. Chinese Science Bulletin 36: 1485-1489.
Zheng, J. Z. et al. 1994. Science in China (Series B) 37: 319-325.
Zhou C. F. et al. 1987. Scientia Agricultura Sinica 20(2): 19-25.
Zhou, M. P. et al. 1999. Jiangsu J. Agric. Sci. 15(1):62-64.
Zhou N. et al. 1993. Jiangsu J. Agric. Sci. 9(2): 22-27.

Fusarium Head Blight - Emerging Issues, an Overview

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In assessing damage from Fusarium head blight (FHB), one must consider about $90 M per year of direct losses, which is compounded by many sorts of risks and indirect losses that are transferred to grain handlers, users, and consumers. Ultimately loss of reputation and loss of market can lead to major socio-economic problems. Use of downgraded grain for feed is tolerated, but not exempt from risks.

FHB has actually gone from the status of rather uncommon fungus in Canada to the status of number one disease of wheat. Perhaps it is timely to ponder the possible reasons for such a dramatic increase, unparalleled in the scientific history of cereal diseases. Trends in rate of FHB will be compared for USA and Canada. Trends in toxin residues in foods will be discussed. The potential health risk for people occupationally exposed and also for infants who are more sensitive to toxins in contaminated food makes FHB a very high research priority.

Hypotheses about the increase of FHB have perhaps been overly focused on climate effects. It is true that rainfall and temperature have increased slightly in the Plains of North America. However, this has to be reconciled with the fact that Quebec was producing wheat of rather good quality, not far from maize crops, from 1970 to 1979, and under rainfall patterns that were quite abundant. Then the FHB problem increased and kept increasing, and yet this was not correlated to a trend of increased rainfall. Among other theories, is often stated that plant genetics is not adequate any more against FHB. Once again, how does one reconcile that with the fact that in Quebec, the cultivars grown in 1970-79 were more FHB-sensitive than those grown nowadays, and yet the crop was generally cleaner. Other factors must be involved besides rainfall and plant genetics.
Within cereal management issues, one can pinpoint some obvious culprits. Soil preparation practices have evolved without enough attention to the impact of these practices on cereal diseases. Rotation crops of the early part of the century were likely not harboring much Fusarium. Seedlots were seldom contaminated. Fertilizer was organic and used sparingly. There was less "mass effect" from vast acreages of maize and wheat, and the biodiversity of microorganisms in the more diversified agro-ecosystem probably provided good competition against the genus Fusarium.

Yet after perusal of the literature available about management issues, there is still no easy explanation about why a fungus group (the genus *Fusarium*) was so rare in our environment for half a century, and why it is now so overabundant, moreover with the most dangerous species (*Fusarium graminearum*) predominating. The question becomes: why is science unable to explain such a blatant trend till now?

More or less "per absurdum", after pondering all possible explanations, one concludes it is time to evaluate the most unpleasant hypothesis, which is that perhaps some very

significant natural selection (or evolutionary) process has been ongoing in the genus Fusarium and perhaps more efficiently in *Fusarium graminearum*. This is unpleasant in the sense it predicts that the problem could get worse. Resistant cultivars are due in the next years; but will the plant breeding progress faster than the fungus, if the natural selection hypothesis was correct?

Despite the funds already invested in FHB research, it is concluded that an all-out effort will be needed, and with special emphasis on basic research, to really understand the behavior of *Fusarium* species and find sustainable ways to reduce the problems. Let us not forget that in many countries, the earliest efforts at combating the cereal rusts were quite unrewarding, due to a lack of basic knowledge. We are, at this point in time, highly vulnerable, and essentially in a situation where we lack basic information about our new enemy.

The socio-economic aspects that relates to FHB and its toxins go far beyond anything we have seen with respect to any plant disease in the past. An integrated approach is needed in the field right now; but to organize the logistics of the fight against FHB, an integrated effort is also needed. Perhaps scientists are going nowhere if there is not behind them a coalition of forces at the national and even at an international level. A number of private and public institutions have a vested interest in knowing more about the problem and about finding sustainable solutions. It is hoped the present meeting will become a milestone in organizing the action of those who want - and can - do something to guarantee that we do not face a further major increase of this problem in the next 10 years, with perhaps fewer and fewer areas where cereals can be grown successfully.

A Consumer Perspective on Food Safety

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The Consumers Association of Canada functions from a set of principles that outline the Rights and Responsibilities of Consumers (Table 1)

Table 1- Rights and Responsibilities of Consumers

Consumer Protection	Right	Responsibility
Safety	Protection from harm	Investigate before use
Information	Facts and information	Ask for information
Choice	Selection	Compare prices and quality
Participation	A role in policy development	Make our needs and expectations known
Compensation	A fair settlement	Insist upon a fair and reasonable deal
Education	Acquire knowledge and develop skills	Ensure that we inform ourselves
Healthy Environment	Now and in the future	Help to build a healthy environment

Food Safety Realities

There are three important realities that need to be considered when attempting to understand the consumer perspective on food safety:

1. We live in a global information age, where what happens around the world is on the front page of our local newspaper. That means that things like BSE- mad cow disease epidemic in the UK and E coli 0157:H7 related illnesses in the United States are presented as if they could have occurred in Canada. In fact, November 1999 is the 50th anniversary of the first mass food 'scare', the Great Cranberry Scare' of 1959 in which cranberries were suspected of being contaminated with dioxin.

2. Most Canadians have a limited understanding of the food production system, including how food is produced, regulated and protected

3. The GE foods 'debate' and consumer demand for unpasteurized products such as cheese and cider are presenting challenges to the existing regulatory system

In my opinion, the Canadian system for food safety should be regarded as one of the most comprehensive in the world. It provides Canadian consumers with ready access to a wide variety of healthy food products year-round. But, since the details of the 'system' are

unfamiliar to most consumers, a large degree of 'trust' is involved. Any factor that affects trust must be dealt with for consumer confidence to be maintained

Food Safety Challenges for the Grains Sector

1. "Commodity' mentality means accountability is difficult to pin-point
2. No direct consumer link; therefore no direct consumer awareness
3. The many layers between the farm and the plate produce many points for possible contamination

Food Safety Issues for the Grains Sector

Canada's Food Guide

- Increased emphasis on consumption of grain and grain-based products will create awareness and questions; is the industry ready to address them?
- Increased consumption could lead to the emergence of previously unforeseen problems
- Is there a pro-active plan for issues management in the grains sector?

'Natural' Products

- Premium prices are charged for 'natural' products. The perception is that they are 'better'; the reality ??
- Are the new marketing systems offered by Farmers markets; internet, mail order, providing consumers with same quality of food products?
- Who is regulating these new systems?

Regulatory Change

- Do pending changes to the variety registration system address the grain industry's long-term interests?
- Making fundamental regulatory changes at a time when there are questions being raised about the current system could result in a crisis in consumer confidence.
- Why are regulations viewed as negative to industrial expansion?

Literacy Levels of Canada

- Literacy is central to the well-being of individual Canadians and Canada as a whole and adult literacy is crucial to the economic performance of industrialized nations. The study "Reading the Future: A Portrait of Literacy in Canada" 1996 redefined literacy as a person's ability to understand and use written information- it is more than the 3 R's!

  The study found that:

  - 22% of Consumers have very low reading skills, but excellent memory and coping strategies. Shopping can be difficult or impossible for these consumers
  - 26% of Consumers require text to be in plain language and clearly

laid out. Long paragraphs will discourage these consumers from reading.

- 53% of Consumers function at the minimum level for literacy. Of this group, 30% are at the minimum level; leaving only 20% of the Canadian population with the skills to understand challenging printed material

What the future holds...

- Public trust is still on the side of our food system in Canada. But this trust is fragile- and being challenged. The grains industry needs to adopt a pro-active issues management strategy, rather than assume that 'no news is good news'
- There is a need for public education activities to broaden awareness and understanding- But they must understand and address literacy concerns
- Considerations of health and safety must be balanced with business and commerce outcomes

1996 Epidemic in Winter Wheat - Aftermath

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The objective of this paper is to demonstrate that fusarium in wheat is an emergency for Ontario and that the emergency registration of FOLICUR is warranted.

The *Fusarium* epidemic of 1996 in Ontario winter wheat was a financial disaster. The Ontario Wheat Producers' Marketing Board (Source: Jim Whitelaw) estimates a 30% yield loss which equates to a 230,000 T loss in volume. At $150/T this equates to a direct loss to producers of $34.5 million. Deoxynivalenol (DON) contamination resulted in quality penalty on about 750,000 T of on average $45/T, another $33.75 million loss to the industry. The crop was extremely difficult to market resulting in an additional $15/T marketing cost totaling about $11.25 million. Another $20-30 million was lost to the industry for buying replacement wheat stocks. The total loss to the *Fusarium* epidemic was well over $100 million.

This epidemic has changed how wheat is marketed in Ontario. The market focus remains primarily food grade. As such the awareness of DON getting into the food chain has increased exponentially and has become a much greater component of marketing and trade. If there is a general fear of DON contamination, Ontario wheat for food is in peril. Market tolerances for DON have been set. The Chicago Board of Trade will only accept up to 5 ppm (DON), while a new tolerance of 0.5 ppm DON has been set in the breakfast cereal markets. The cereal markets no longer make advanced purchase contracts, but the markets have changed to post-harvest sales.

The marketers of Ontario wheat experience a new reluctance to service export customers, because of the liability associate with DON. Furthermore, DON is a special problem because there are no markets for process by-products which contain concentrated levels of DON.

Before 1996, DON problems were handled largely by blending across the province. A reluctance to blend DON contaminated wheat is growing, and if there is any suspicion of DON contamination markets will move to checking individual loads at the source.

A field level study of winter wheat samples taken at harvest across Ontario over three years including 1996 (Fig. 1) showed that environment and wheat variety were the main contributors to epidemics. Other agronomic factors such as crop history and tillage practices were statistically significant but minor contributors. The main approach that Ontario has taken to manage this disease and DON is to develop resistant varieties. This task, while noble and important, is not likely to come to fruition in the near future. In the meantime, other tools are recommended in an integrated approach. Ontario is recommending good crop rotation, selecting more tolerant varieties (note that there are no resistant commercial cultivars available), forecasting epidemics, treating with fungicides, modified harvesting and cleaning strategies, and selective marketing.

Most of the data from around the world and recently from North America suggest that the fungicide FOLICUR (tebuconazole) consistently reduces DON levels by about 50%. If this fungicide had been applied during the epidemic of 1996, the 750,000 T that

averaged about 8-10 ppm DON could have been marketed through the Chicago Board of Trade, falling below its 5-ppm tolerance. The epidemic would have been $37.5 million less of a problem.

Since 1996, Ontario has experienced DON contamination at levels above 1 ppm in scattered, localized areas each year across the province, all related to weather factors. In 1999, corn producers in 5 counties experienced the worst epidemic of Fusarium since 1986, with levels of 5-10 ppm DON showing up in their corn destined for hog production. These same counties are where most of the winter wheat is produced in Ontario. Thus the inoculum potential for next year is large.

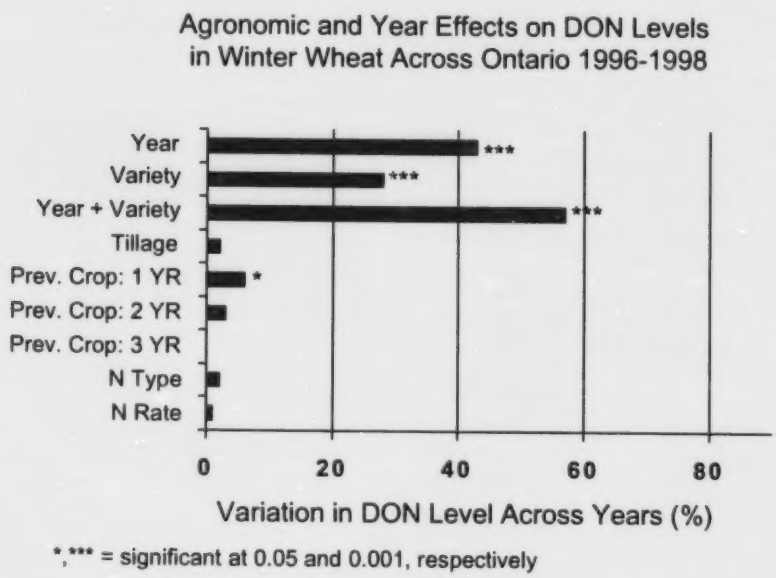

Figure 1. Portion of the variation in deoxynivalenol content measured in winter wheat samples collected across Ontario explained by various agronomic factors in three years of sampling. (N = nitrogen fertilizer)

With the change in marketing of wheat because of: the threat of DON, the low commodity prices to begin with, the fragile nature of the Ontario wheat business, the huge inoculum potential in the field, it only makes sense to declare an emergency against *Fusarium*. While FOLICUR will not solve the problem, it will help producers of wheat in Ontario mitigate their losses, and maintain a viable outlet for their crop. An emergency registration for FOLICUR is easily justifiable.

Developing Threat of FHB to Saskatchewan and Alberta

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Fusarium head blight (FHB) is a disease of small grain cereals affecting the yield, grade, and quality as well as contaminating the grain with mycotoxins. Three species of *Fusarium*, *F. graminearum* Schwabe, *F. culmorum* (W.G. Smith) Sacc. and *F. avenaceum* (Corda ex Fr.) Sacc. are important as causal agents of the disease. In North America, *F. graminearum* is the primary FHB pathogen. Although both *F. culmorum* and *F. avenaceum* can and do cause FHB in Canada, their importance here is limited.

Fusarium graminearum is not new to the grasslands of North America. It has been present in the upper midwest US since at least 1900. In western Canada, *F. graminearum* was detected as early as 1923 on corn stubble in Winnipeg (Bisby and Bailey, 1923). Surveys by Gordon and others established that this pathogen was rare in western Canada (Gordon, 1944, 1952, 1954, 1956; Gordon et al, 1948; Greaney and Machacek, 1942; Machacek et al 1951). In 1984 it was found at high levels in 2 samples of Manitoba wheat. The mycotoxin deoxynivalenol (DON) was also present (Clear and Abramson, 1986). At that time the conventional wisdom was that the environmental conditions of the prairies, notably the amount of precipitation, and the small amount of acreage in western Canada planted to corn (corn stubble was considered an important factor in the previous epidemics in the USA and Ontario because it is an excellent source of inoculum for the disease) was not suitable for this organism to become an important cereal pathogen.

Since 1984, *F. graminearum* has been detected from an increasing area of western Canada. By 1993 it was well established in Manitoba, and detectable in a few fields in southeastern Saskatchewan. By 1998 it was well established in eastern Saskatchewan and present in all CD's in Alberta. To date, most of the locations in western Canada where *F. graminearum* has been detected are within the black soil zone, which is also the area of highest moisture. In areas where *F. graminearum* has become established, the frequency with which *F. culmorum* and *F. avenaceum* are recovered from fusarium damaged kernels (FDK) has declined sharply. In addition, increasing dominance of *F. graminearum* has coincided with increased FHB. In recent years, between 77% and 92% of FDK in Manitoba were infected by *F. graminearum*. Since 1997, *F. graminearum* has been the dominant FHB pathogen in Saskatchewan as well.

Many factors have influenced the recent spread of *F. graminearum* and its emergence as a serious pathogen of cereal crops on the eastern Canadian prairies. Precipitation during anthesis (July) is considered an important factor in disease development. Precipitation levels recorded in southern Manitoba since 1984 are not unique to the prairies. Many areas of Alberta and Saskatchewan have recorded similar precipitation during those years. What is unique is the development of *F. graminearum* to epidemic levels in southern Manitoba and southeastern Saskatchewan. In areas where FHB is a serious problem,

there has been a steady increase in recovery of *F. graminearum* from seed over the last 15 years. Even areas now heavily infected were once virtually free of this pathogen. There is a risk that this pathogen will become endemic in more areas of western Canada. However, temperature may be a factor limiting disease levels in the western prairies. It is considered a primary factor in deciding which *Fusarium* species will predominate in an area (Cook 1981), and infections require longer to develop at lower temperatures. Average daily means in July for many areas in the western prairies are 3 to 4 °C below those in the worst affected areas of the eastern prairies. However, the longer periods of daylight in the more northern locations may compensate for the lower night time temperatures. Perhaps the average hourly temperature during July is not as different as is the average of the daily maximum and minimum.

Infected seed may be serving as a long distance dispersal mechanism. Infected seed is considered an important dispersal mechanism for many fungi which cause plant diseases, and may well be serving this same function for the spread of *F. graminearum*. The use of clean seed and the application of a seed treatment effective against *Fusarium* species is recommended to both control seedling blight and, in areas where it is rare or absent, to perhaps delay the introduction of *F. graminearum* into fields free of this pathogen. In an effort to deal with this risk, Alberta has placed *F. graminearum* on their list of declared pests. This gives the power of *F. graminearum* prevention and/or control to the municipality. Local authorities have the option to enforce, provide a warning, or do nothing.

Another possible factor contributing to the recent rise in importance of *F. graminearum* is changes to the pathogen. Perhaps *F. graminearum* has adapted to prairie growing conditions. In 1952 Gordon reported that ascospores of *F. graminearum* in Manitoba matured too late to be of much importance to FHB. This may no longer be true, as severe disease levels have been found on cereals flowering in late June and early July.

Although *F. graminearum* was found to be capable of causing FHB in essentially all CD's in western Canada, it is still uncertain if it will ever be an important pathogen of cereals in Alberta or western Saskatchewan. Climatic differences between the eastern and western prairies, notably in temperature and moisture, may serve to contain the spread or limit the damage caused by this species. However, in areas where levels of *F. graminearum* are presently very low, it would be prudent to adopt control measures now that might reduce future losses. Until such time as resistant varieties become available, only modest success in dealing with this pathogen can be expected. However, the economic impact of *F. graminearum* is such that even a modest success will more than repay the cost of achieving it.

References

Bisby and Bailey. 1923. Page 33 In Fourth Annual Report of the Survey of the prevalence of plant diseases in the Dominion of Canada, Ottawa, Ont.

Clear, R.M. and D. Abramson. 1986. Can. Plant Dis. Surv. 66(1): 9-11.

Cook, R.J. 1981. Pages 39-52 in: *Fusarium* Diseases, Biology, and Taxonomy. P.E. Nelson, T.A. Toussoun, and R.J. Cook, eds. Pennsylvania State University Press, University Park.

Gordon, W.L. 1944. Canadian J. of Research 22: 282-286.

Gordon, W.L. 1952. Can. J. Bot. 30: 209-251.

Gordon, W.L. 1954. Can. J. Bot. 32: 622-629.

Gordon, W.L. 1956. Can. J. Bot. 34: 833-846.

Gordon, W.L., J.E. Machacek and W.A.F. Hagborg. 1948. Can. Plant Dis. Surv. 28: 1.

Greaney, F.J. and J.E. Machacek. 1942. Sci. Agric. 22: 419-437.

Machacek, J.E., W.J. Cherewick, H.W. Mead, and W.C. Broadfoot. 1951. Sci. Agric. 31: 193-206.

Folicur Emergency Use – 1999

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Bayer Inc., Toronto, Ontario

Introduction:
In the following presentation I will discuss the following topics as they relate to the issues of 1999 and the Emergency Use granted to Bayer Inc. for the limited sales of the fungicide Folicur in Western Canada, particularly Manitoba and Saskatchewan.

1. World Cereal Production
2. Canadian and US Issues
3. Folicur (tebuconazole) introduction
4. US Section 18
5. Canadian Emergency use & results
6. What about 2000?

World Cereal Production

The following slide shows a summary of world estimated grain production areas since 1996. The former USSR has the most land committed to grain production with between 46 and 48 million hectares. The US, China and India follow this with a production area of 25-30 million hectares. The EU and Canada have the smallest area committed to grain production with 11-15 million hectares or about 20 million acres.

Interestingly when yields are compared, the results look much different. The former USSR, which has the largest area committed to cereal production, has the lowest yields. The USSR also has the record for the lowest outside inputs including fertilizers and crop protection chemicals. The EU on the other hand, which had one of the lowest areas committed to cereal production, has the highest yield of any country. This is due to the high input of fertilizers and crop protection chemicals including herbicides and fungicides. The EU also has committed government support programs that encourage and pay for high production on limited crop acres. Canada and the US produce mainly without government programs with similar respectable yield responses.

Canadian and US Issues
The following two charts show the trend of US import and export of wheat since 1994. Interestingly the US is not noted as an export country but they do export nearly half of their annual production of about 31-35 million metric tons of wheat. The other half of the US production is used for internal consumption and animal feed. Most interesting for Canadians is the import market in the US. This market has been growing since 1994. This is the year of the Fusarium outbreak in the US that lowered wheat quality and millers were looking elsewhere for high quality wheat without the FHB issues.

This slide shows in the increasing value to Canada for producing quality wheat. Imports to the US have increased by nearly $50 million dollars (US) each year since 1994. After

the experience in 1994 the growers in the northern states asked and received an Emergency Use label for the use of Folicur to increase wheat quality and reduce the movement of Canadian wheat into previously held US markets. If Canada is to continue to grow this export market, we must be able to supply this high quality wheat.

Canada is a major export country and in order to compete with the EU, Australia and Argentina for market share, we have to supply high quality wheat.

Folicur (tebuconazole) introduction

As outlined in the previous overheads, producing high quality wheat is imperative to Canada remaining an exporter of choice. Folicur was selected as the fungicide of choice in the EU and the US to reduce the impact of Fusarium. The response of Fusarium to Folicur is based on rate. Folicur has the following profile:

- ✓ Common name - tebuconazole
- ✓ Chemical name - (1-(4-Chlorophenyl)-4,4-dimethyl-3-[1,2,4]-triazol-1-ylmethylpentan-3-ol)
- ✓ 3rd generation azole/triazole systemic fungicide
- ✓ Also sold as Raxil seed treatment
- ✓ Acute oral tox. LD50: 1700mg/kg - female rat, Acute dermal tox. LD50: >5000mg/kg
- ✓ No mutagenic, carcinogenic or teratogenic effects
- ✓ No skin or eye irritation, not a skin sensitizer
- ✓ Tebuconazole is non-toxic to birds, earthworms, soil microflora
- ✓ Immobile in soil; no leaching
- ✓ No risk to fish, their diet, or algae
- ✓ Safe to bees and other beneficials
- ✓ Systemic mode of action (acropetal)
- ✓ Does not accumulate in apical leaf or shoot regions, all plant parts protected
- ✓ Penetrates rapidly into plant and is rainfast in 2 - 4 hours
- ✓ Effective for about 14 days after application

Folicur has a good environmental profile. However, the biggest issue faced by PMRA is that Folicur does persist in the soil and is broken down slowly by soil microbes.

US Section 18

As mentioned above the US received an Emergency Use (Section 18) beginning in 1994. The emergency was justified on the basis of reduced wheat quality and increased DON (deoxynivalenol) levels in US wheat and the increased value of Canadian imports. Folicur has been shown to reduce Fusarium and DON levels. The US anticipates renewal of the wheat Section 18 in the states of ND, SD, MN, MI, OR, ID, WA and CA for 2000. The Folicur registration (Section 3) was submitted to EPA in July 97 with an expected full registration in May or June 2000. The current US use rate is 4 oz/A (125 g ai/ha), however, the US is planning for rate increase to 6 oz/A (190 g ai/ha) for increased FHB control and reduced DON levels in 2Q – 2001.

In the US the minor use program or IR-4 seeking a Folicur registration for barley.

Folicur has been registered in the EU for many years. A table of the use rates in other countries with high demand registration systems is listed below:

Country	Trade Name	Formulation	Rate A.I.	Mixture
Germany	Folicur	250 EW	250 g ai/ha	No
France	Horizon	250 EW	250 g ai/ha	No
UK	Folicur	250 EW	250 g ai/ha	No
New Zealand	Folicur	250 EW	250 g ai/ha	No

Important in the above chart is the rate. In Canada we are applying for one-half the rate currently registered in the EU countries. However, as all ready mentioned, these countries also have strong government support systems that make high inputs possible.

Other countries also have Folicur registered, but these countries usually rely on an EU or US EPA registration for approval. The rates in these countries are generally lower, mostly because of input costs.

Country	Trade Name	Formulation	Rate A.I.	Mixture
Hungary	Folicur BT	225 EC	125 g ai/ha	Yes
Poland	Folicur BT	225 EC	125 g ai/ha	Yes
Czech Rep	Folicur BT	225 EC	125 g ai/ha	Yes
Slovenia	Folicur BT	225 EC	125 g ai/ha	Yes
Croatia	Folicur BT	225 EC	125 g ai/ha	Yes
Serbia	Folicur BT	225 EC	125 g ai/ha	Yes
Russia	Folicur	250 EC	250 g ai/ha	No
Argentina	Folicur	250 EW	125-187 g ai/ha	No
Argentina	Folicur	430 SC	150-193 g ai/ha	No
Argentina	Coloso	300 EC	123-180 g ai/ha	Yes

Canadian Emergency use & results

The Emergency Use was requested by growers and supported by provincial experts in Manitoba and Saskatchewan. The moist spring conditions set the stage for rapid disease development. The US EPA approved Section 18's in the northern states. Growers in Canada reviewed with PMRA the alternative products labeled for use in Canada. They found that Folicur consistently reduced DON levels and consistently increased yields from results in provincial tests and US data. In late May the Emergency Use in Canada was received. The Emergency Use period was from June through September 99.

The conditions of the PMRA emergency approval was based on the strong support from provincial experts. PMRA asked the growers be made fully aware of strengths and weaknesses of Folicur and that the 125 g ai/ha rate would only give FHB suppression. Farmers were to assess economics before spraying and only apply when a yield potential

and economic return could be expected. Farmers were also encouraged only to spray when Fusarium risk was high. A special web site was introduced by Manitoba Ag to give a weekly updates on the spread of the disease and conditions where the disease could be expected. Only wheat varieties that had some natural FHB resistance were allowed to be sprayed.

Bayer also agreed to give training for the safe use of Folicur. This material was given to all applicators. Special training meetings were held in key areas where the Fusarium outbreak was occurring or expected. Aerial applicator training meetings were also held on best application techniques. Bayer also held an information phone-in program, where growers could call a toll free number and hear a panel discuss Fusarium, how the disease spreads and how to use Folicur in the most effective manner. Bayer also agreed the product would only be sold on wheat and all unused Folicur would be returned at season end.

The results of the Folicur emergency were encouraging. A few Folicur fields were taken to yield. Yields were from combine monitors, weigh wagons or elevators. Sample bags were also provided for DON level evaluation. The Board of Grain evaluated the samples for percent Fusarium kernels. All varieties used in the yield trials contained some natural FHB resistance.

In general the number of Fusarium infected kernels was reduced by half with the addition of Folicur. The fields with the higher disease symptoms showed more FHB reduction. Yields were also increased. Generally the increase in yield was about 10 bu/A, but varied due to FHB infection level. Seeds were also evaluated for DON levels and were found to have decreased DON levels by at least 50% compared to the untreated controls. In every sample where Folicur was used the DON was reduced below 1ppm, which is considered the new cutoff for grain to be considered clean.

What about 2000?

Bayer has met with PMRA regarding the full registration for Folicur. Bayer has submitted to PMRA a list of the current US studies available for review. PMRA is reviewing the list of studies and will make a request to Bayer for those needed to make a full assessment of Folicur for Canada. However, the provinces must make their needs known to PMRA. Each province needs to document current Fusarium infestation levels and need for Folicur in 2000. The main question for PMRA - Is the emergency real?

The Regulatory Challenge Of Emergency Use Requests

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The purpose of this talk is to give some understanding as to how emergency use requests are handled with respect to making disease control products such as Folicur available within a relatively short time. This has particular relevance to Fusarium Head Blight, which is a chronic and expanding problem but with only sporadic epidemics - does this fit an emergency by the Agency's definition?

Current emergency criteria, as per Regulatory Directive 94-05, include:

1. a pest (disease) outbreak is expected to occur that can cause significant economic, environmental or health problems
2. no products are registered in Canada for control of the pest
3. no alternative control method is available
4. this is not intended as an option for ongoing pest problems or the ongoing need for a registered product to fill a void ie. not intended to bypass the regular review process

There are also practical considerations: the Minister has authority to register a product for the proposed use for a period not exceeding one year and the product must be safe and efficacious for this use, with no major safety concerns which would preclude expansion to new crops or additional sprays to control a new disease. Registration may be limited to the area where this product is critical to protect a crop. If applicable, there must be time to establish an MRL for food use (can take over a year, but can be shorter if an Interim Marketing Authority is accepted), and the application requires the written support of a provincial or federal agency (sponsor) and consent of the company. These requests are most effective if they are for new use of registered or reviewed product or at least a product already under evaluation.

There is an expectation that agencies in both Canada and the US can allow emergency use of pesticides in the same timeframe if there is the same disease scenario, however the two countries have different mechanics for handling emergency use requests. One key difference is that the US EPA grants an *exemption* from registration whereas PMRA is obliged to grant *registration* of a product for the emergency use. The EPA establishes a time-limited tolerance, if needed, to cover expected residues, whereas PMRA establishes a regular MRL after standard full data review and gazetting. If there is insufficient time for a full EPA review, the states may issue a crisis exemption for up to 15 days (registered products only) whereas the provinces lack this authority. This type of exemption is rare, and a temporary tolerance is still established, after product use but prior to sale of crop. Finally, the EPA has a target date of 50 days and an allocated review team for emergency use requests, whereas PMRA has no specified timeline or dedicated review staff. To summarise, emergency use requests are one task which is not harmonized at this point, which often results in different response times from the two agencies.

How does Fusarium Head Blight fit as an emergency use request?
It meets criterion 1 because of documented damage to grain quality, mycotoxin concerns and downgrading costs, in addition to concerns with spread of the pathogen to regions previously not affected by FHB. Another product (Bravo) is registered for this disease but apparently has not been accepted by growers as a viable option. Other control measures (tolerant or resistant varieties) are not expected for a few years. Rotation is of limited use in areas where corn/wheat are routinely grown. Is FHB an emergency or an ongoing pest problem??? The challenge is to determine if exceptional circumstances will result in major damage this use season, yet know this in time to register and make available a suitable fungicide product within the narrow window for application (flowering).

Other options for registration to keep in mind include URMULE for small acreage or specialised new uses of registered products. It appears that this is still underused as a way of avoiding a need for last-minute emergency use requests. Joint reviews with the EPA may be used for new active ingredients with reduced risk status - still long term but can result in shorter registration timeline and co-ordinated introduction of product into both countries. Finally, PMRA can manage *occasional* requests for expedited review of a regular submission to meet the use season - this depends on workload and maturity of the review.

We expect more success where commodity groups are co-ordinated across the border, anticipate North American pest/disease problems and get the company, extension staff and Agencies working on this early in process (eg. canola council efforts). This avoids 'catch-up' pressure, usually on Canada, and trade issues with residue tolerances or treated seed. Prioritizing product needs for a crop avoids crying wolf or having products for different disease problems competing for attention and review time in the registration system. This Workshop focuses on one disease of cereals but the collaborative approach is a step in right direction.

Common *Fusarium* Mycotoxins and their Detection

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Of the many metabolites *Fusarium* fungi can produce, only about a dozen show toxicity by the oral route of ingestion, and occur as natural contaminants of crops. This group includes deoxynivalenol (DON), 3-acetylDON, 15-acetylDON, nivalenol, fusarenone, diacetoxyscirpenol, T-2 toxin, HT-2 toxin, moniliformin and fumonisin B_1. The fungal estrogen zearalenone is often included in this group.

Presence of *Fusarium* toxins decreases the market value of cereal crops. In 1998, cereals accounted for 34% of the value of the total crop sector nationally ($13.8B) and 47% of the total crop sector in the Prairies ($8.3B). The detrimental effects of *Fusarium* toxins in cereal-based feeds are limited mainly to swine. In 1998, swine accounted for 16% of the value of the total livestock sector nationally ($14.1B), and 20% of the total livestock sector in Ontario and Quebec ($6.6B).

Commercial enzyme immunoassay kits are available to screen commodities for DON, T-2 toxin, fumonisin B_1 and zearalenone. Gas chromatography (with or without mass spectrometry) is commonly used to estimate other *Fusarium* toxins except for moniliformin, which is usually assayed by liquid chromatography. Thin-layer chromatography is quick and economical, although generally not as accurate as other methods. But studies of aflatoxins in peanuts have shown that only 2% of the testing error is due to the analytical method, while 98% of the testing error is due to sampling and subsampling problems. Producers and handlers of grain are advised to employ well-validated sampling protocols to obtain representative samples of grain for mycotoxin testing, and to ensure valid results.

Epidemiology of Fusarium Head Blight in Eastern Canada- When and Where

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Over the past 8 years, my research has been attempting to answer basic questions about the temporal and spatial aspects of the perfect stage, *Gibberella zeae* under field conditions. For example, when are perithecia produced and what are the environmental conditions required for formation of perithecia and maturation of ascospores? In 1997 and 1998 in L'Acadie, Quebec, we inoculated 2 X 2 m microplots of spring wheat cultivar Roblin with macroconidia of *F. graminearum*. In the fall, the infected wheat was cut and 10% was returned to the plot, along with other samples of straw and spikelets placed in small screen sachets. Every month from October to July, samples were recovered and the maturity of perithecia on the debris was assessed with a 1-6 rating scale. In 1998, a 10 X 10 plot of wheat was inoculated at Macdonald Campus and all the residue was left on the field in the fall. Samples were taken every two weeks from September 1997 to August, 1998. Debris moisture, soil and air temperature and rainfall were continually monitored. At all sites, mature perithecia were formed in September and October. At the Macdonald site in December, 1998, 85% of the perithecia were mature with ascospores. The ascospores present in the perithecia in December and the following spring had an unusual morphology. The septations were constricted, giving the spores a monilioid appearance. In some of the ascospores, intact cells fragmented from the main spore, and some of the spores had germinated. The following spring, 70% of the overwintered ascospores were viable. After snow melt in March and April, the maturity ratings increased, but decreased in May as a new crop of perithecia were being formed. This new crop of perithecia matured in late June- early July. Ascospores were also trapped over the plots with rotorod volumetric samplers. In 1999, over 16 spore release events with concentrations > 100 ascospores/m^3 were recorded from the Macdonald plot from June 1 to August 30. Most events consisted of 1-3 nights of release, with one 8-night event. Ascospore release began 0-7 days after rainfall > 1 mm. Only 10% of the releases occurred on the same day as rainfall. The highest concentrations (> 1000 ascospores/m^3) were recorded in releases 1-3 days after rainfall, with decreasing spore concentrations as the time after rainfall increased. To determine how far ascospores could travel from inoculum sources, transects of five spore traps each were set up in 1998 and 1999, in a downwind direction from the inoculated plot. In 1998, the transects were 25 m long, 50 m long in 1999. In 1999, about half of the gradients showed a significant fit with the exponential model. With spore releases > 100 ascospores/m^3, the average D_{50} (distance at which spores declined 50% from source) was 21 ± 2 m, while the D_{90} was 70 ± 5 m. For release events < 100 ascospores/m^3, the D_{50} was 16 ± 8 m, and the D_{90} was 52 ± 28 m. These results show that there are two crops of perithecia (fall and spring) formed under Quebec conditions in 1997-1999, however the spring and fall of these years were abnormally warm. Ascospores can survive in the perithecia over the winter, but the epidemiological consequences of this inoculum are unknown. There were more releases from naturally overwintered inoculum than from non-overwintered laboratory-raised

inoculum in previous experiments. Inoculum on overwintered debris can release ascospores many times from June-August, suggesting a diverse population of perithecia with varying maturities. Inoculum may be present throughout the growing season, if moisture is favorable for perithecial formation and maturity. If infection can take place through the soft dough stage, as previous experiments have indicated, wheat may be at risk through the beginning of August. Finally, the spores travel much further from larger areas of inoculum than predicted with previous experiments with small 1 x 1 m plots measuring seed infection.

Fusarium Head Blight, A Producer's View

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Producer comments centered on the economics of FHB as they affect cropping budgets, agronomic methods of accommodating FHB in a cropping plan, and examples of opportunity costs associated with FHB. The paper concluded with producer needs re FHB in a perfect world.

In reporting losses to FHB, preharvest surveys and post-harvest sample analysis are used to estimate costs to the industry. Such averaging methods often mask the real impact on a single farming operation. Studying harvested grain samples does not reflect economic loss because producers make every effort to remove damaged kernels during harvest.

Producers consider yield in dollars/acre. Factors affecting dollar returns include grain yield, protein content, grade, and special marketing opportunities. As crop is stressed due to FHB, grain yield drops but protein content often climbs. Presence of FHB often precludes participation in quality dependent premium markets.

Producer agronomic considerations include rotations, crop kind and variety selection, and use of control products. FHB has resulted in lengthening of cereal rotations, especially corn, barley, and wheat. Present experience suggests that producers avoid wheat varieties with a beard. This excludes durum wheat, CPS wheat, and most US bread wheat varieties. There is a wide variation in susceptibility of hard wheat varieties to FHB under modest infections. At high rates of infection, variety differentiation has less meaning.

Control initially consisted of seed treatment, but this is not widely used at present. High quality, well cleaned seed is the first means of control. Foliar sprays have been economic in recent years, but it is unclear whether the benefit is from FHB control, or from control of other leaf diseases. Experience suggests that a healthy plant is better able to resist FHB infection.

Opportunity costs caused by FHB include restrictions on rotations, restrictions on classes of wheat that can be grown, restricted variety choices, and exclusion from special marketing opportunities.

Producer needs re FHB include a more definitive variety evaluation, an early warning system similar to potato late blight, and control recommendations linked to threshold levels and economic models.

There is a wealth of on-farm producer experience and the FHB initiative could profit from continued producer - researcher communication.

Fusarium Head Blight: Effect on Wheat Milling and End-Product Quality

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Introduction

Fusarium head blight has been a serious concern in eastern Canada since 1980, and has been evident in the eastern part of western Canada (primarily *F. graminearum* Schwabe) since 1984. Fusarium head blight outbreaks are a health concern because of mycotoxins in fusarium-damaged (FD) grain. Initially wheat grade standards were set primarily on a food safety basis. Recently there has been increasing awareness that FD can have quality implications at levels below the maximum tolerated for safety (Pomeranz et al. 1990 and references therein). At the 1996 Regional Fusarium/Scab Forum in Winnipeg Dexter et al (1996a) concluded that the effects of FD on wheat milling, baking and pasta-making performance need to be considered when establishing FD limits for wheat milling grades. This report presents an update of recent Canadian Grain Commission (CGC) research on effects of FD on wheat processing characteristics, and implications for FD tolerances in wheat milling grades.

Quality Effects of Fusarium Damage and Impact on Tolerances in Canadian Wheat Grades

Research conducted by the CGC at the Grain Research Laboratory (GRL) has demonstrated that FD has deleterious effects on wheat milling and end-product quality (Dexter et al. 1996b, 1997). Typical results are shown for Grandin (American red spring variety) and Roblin (Canada Western Red Spring variety) from the 1994 western Canadian wheat harvest (Table 1).

Table 1. Effect of fusarium damage on wheat processing quality[1].

Property	Grandin		Roblin	
	CL	AS	CL	AS
Wheat:				
Test weight, kg/hL	79.0	76.7	77.9	75.0
FD, %	1.5	7.3	0.2	5.9
Flour yield, %	75.0	74.7	74.1	73.5
Flour:				
Protein, %	13.2	13.1	14.5	14.4
Grade color, Kent-Jones units	0.1	1.2	-0.1	0.9
Farinograph:				
Absorption, %	62.4	62.0	62.5	62.0
Develop. Time, min	4 ½	4	6 ½	5
Stability, min	8	7	9 ½	9
Bread (remix process)				
Absorption, %	60	58	61	61
Remix time, min	2.0	2.3	1.2	1.1
Loaf volume, cc	920	855	690	520
Strength index, %	106	99	72	55

[1] "CL" = FD removed by hand-picking; "AS" = as is without removing FD kernels.
Source: Dexter et al. 1996b.

Conclusions of GRL studies, which include quality evaluation of FD samples of hard red spring varieties from the Canada Western Extra Strong (CWES), Canada Western Red Spring (CWRW), Canada Prairie Spring Red (CPSR) classes, and Canada Western Amber Durum (CWAD) wheat varieties, can be summarized as follows:

- FD reduces test weight. Flour or semolina yield is only moderately affected, but flour or semolina color deteriorates significantly.
- Physical dough properties as assessed by farinograph and mixograph curves indicate a moderate loss of dough strength.
- For common wheat, baking quality was affected more than expected from the moderate loss of dough strength. Effects on baking quality were most pronounced for long fermentation processes. Some varieties exhibited very unsatisfactory baking quality even when FD kernels were removed by hand-picking (see data for Roblin in Table 1).
- For durum wheat, FD at relatively low levels made pasta become duller and more red. Pasta cooking quality did not appear to be affected.
- FD wheat contained a lower proportion of high molecular weight glutenin protein aggregates, which are key factors determining dough strength and baking performance.

The lower proportion of high molecular weight glutenin aggregates in FD wheat was postulated to be due to immaturity associated with FD, or to the effect of fungal proteolytic enzymes. The latter appeared most likely as the primary cause, because

degradation of protein by proteases during fermentation would explain the poor baking performance of FD wheat for long fermentation processes. Nightingale et al (1999) showed that when dough was rested up to two hours, the effect of FD on dough consistency and resistance to extension, as measured by farinograph and extensigraph curves, became increasingly obvious. They also used size exclusion high-performance liquid chromatography to follow hydrolysis of wheat storage protein by proteolytic enzymes from FD wheat, and from pure cultures of *F. graminearum*. They concluded that loss of dough functionality and loaf volume potential of FD wheat was primarily due to storage protein degradation by fungal proteases.

Table 2. Tolerances for FD (%) in western Canadian wheat grades.

Glass and grade	Before August 1, 1999	AfterAugust 1, 1999
No 1 CWRS	0.25	0.25
No 2 CWRS	**2.0**	**1.0**
No 3 CWRS	2.0	2.0
No 1 CWES	**2.0**	**1.0**
No 2 CWES	**2.0**	**1.0**
No 1 and No 2 CWAD	0.5	0.5
No 3 and No 4 CWAD	2.0	2.0
All grades of other classes	2.0	2.0

The maximum tolerance for No 2 CWRS and for No 1 and No 2 CWES was lowered from 2% to 1% effective August 1, 1999 because of CGC research results, and evidence that FD was associated with weakness in some export shipments (Table 2). The existing strict tolerances of 0.25% in No 1 CWRS and 0.5% in No 1 and 2 CWAD were deemed sufficient to protect processing quality of those premium grades.

FD tolerances for other wheat milling grades were left at 2% because of lack of evidence that FD tolerances impeded marketing. In SE Asia noodles are often the primary end-product of Canadian wheat. Little is known about the effects of FD on Asian noodle quality. Therefore, the GRL undertook an investigation to document the effects, with the possibility of eventually reconsidering FD tolerances for grades intended for high quality noodles.

FD samples of Canada Western Red Winter (CWRW) from the 1998 Canadian Grain Commission harvest were available for preliminary evaluation of the impact of FD on noodle properties. Nine samples ranging from 0.5% to 9% FD were prepared. Results from patent flour processing of three of the composites representing the full FD range are shown in Table 3.

Milling and physical dough properties were consistent with results of previous studies summarized earlier. Flour color deteriorated with increasing level of FD (a shift of 0.5 K-J units is readily observable by eye, and equivalent to the normal darkening seen as flour extraction rate increases about 1%). Micro-mixograph curves indicated a moderate loss of dough strength.

Table 3. Effect of FD on patent flour color and noodle-making quality of CWRW wheat.

Property	0.5% FD	3.7% FD	9.6% FD
Wheat:			
Test weight, kg/hL	78.5	77.6	76.1
Flour yield, %	60.0	60.0	60.0
Patent flour:			
Protein, %	9.2	9.4	9.2
Grade color, K-J	-2.2	-1.5	-0.3
Mixograph:			
Peak resistance, units	43.2	38.4	37.2
Bandwidth (8 min), units	12.7	10.4	10.5
Work input (8 min), units	270	239	244
White salted noodles:			
Color (24 hr)			
L*	75.1	73.9	73.2
a*	3.4	3.8	3.6
b*	29.4	29.1	27.0
Cooking quality:			
Recovery, %	26.0	21.6	20.2
Max. cutting stress, g/mm^2	17.5	15.6	15.1
Kansui noodles:			
Color (24 hr)			
L*	73.5	71.4	70.4
a*	0.7	1.4	1.8
b*	34.6	34.5	32.5
Cooking quality:			
Recovery, %	25.3	24.2	20.8
Max. cutting stress, g/mm^2	16.6	15.1	14.2

The characteristics of both white salted (1% NaCl) and alkaline noodles (1% *kansui*; 9:1 Na_3CO_3 and K_2CO_3) were influenced by FD. As FD increased noodles became darker (lower L*) and less yellow (lower b*). For alkaline noodles, FD also imparted an undesirable redness (higher a*). For both types of noodles dark specks became more numerous and more pronounced. Cooked noodle resilience (recovery from compression) and firmness (maximum cutting stress) declined.

Results from these studies have confirmed that FD can have serious quality implications for Asian noodles. These studies are continuing using other wheat samples, with

particular emphasis on the CPS-White class, which is targeted primarily for noodle-making.

Conclusions

Food safety and loss of agricultural productivity remain the primary concerns with FD wheat. However, impact of FD on processing expectations for premium quality bread is apparent at levels below minimum food safety standards. Therefore,, maximum FD tolerances in some Canadian wheat grades were lowered in 1999. Preliminary results indicate that FD tolerances in wheat grades intended for premium noodle markets may need to be reevaluated due to poor noodle color and inferior noodle texture associated with FD.

Acknowledgments

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References

Dexter, J.E., Clear, R.M., Preston, K.R., Marchylo, B.A. and Clarke, J.M. 1996a. Fusarium head blight: Wheat processing implications. Pages 12-17 in: Proc. 1996 Regional Fusarium/Scab Forum, October, 1996, Winnipeg, MB. R.M. Clear, ed. Canadian Grain Commission, Winnipeg, MB.

Dexter, J.E., Clear, R.M., and Preston, K.R. 1996b. Fusarium head blight: Effect on the milling and baking of some Canadian wheats. Cereal Chem. 73: 695-701.

Dexter, J.E., Marchylo, B.A., Clear, R.M. and Clarke, J.M. 1997. Fusarium head blight: Effect on semolina and pasta-making quality of durum wheat. Cereal Chem. 74:519-525.

Nightingale, M.J., Marchylo, B.A., Clear, R.M., Dexter, J.E. and Preston, K.R. 1999. Fusarium head blight: Effect of fungal proteases on wheat storage proteins. Cereal Chem. 76:150-158.

Pomeranz, Y., Bechtel, D.B., Sauer, D.B., and Seitz, L.M. 1990. Fusarium head blight (scab) in cereal grains. Pages 373-433 in: Advances in Cereal Science and Technology Volume X. Y. Pomeranz, ed. American Association of Cereal Chemists, St. Paul, MN.

Utilization of Vomitoxin Contaminated Grain as Animal Feed

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Vomitoxin contaminated grain can be utilized safely in cattle and poultry rations. Cattle fed a complete ration containing 10 ppm vomitoxin for 18 weeks showed no reduced feed intake. Poultry fed a complete ration containing 100 ppm vomitoxin for 1 week showed no reduced feed intake. Vomitoxin contaminated grain can not be utilized in swine rations. A complete swine ration containing greater than 1 ppm will result in reduced feed intake. A significant percentage of vomitoxin contaminated grains will also contain the mycotoxin zearalenone. Complete swine rations containing 1-5 ppm zearalenone will cause vulvovaginitis in gilts. Complete swine rations containing 3-10 ppm zearalenone will cause anestrus and/or pseudopregnancy in sows. Cattle fed a complete ration containing 12.5 ppm zearalenone resulted in a reduced conception rates in heifers. Cattle fed a complete ration containing 50 ppm zearalenone resulted in a reduced conception rate in cows. Use of a grain probe is required to obtain an adequate grain sample for mycotoxin analysis. A minimum of 10 samples should be obtained from various areas of the grain bin and then be composited into a single sample of approximately 0.5 kg. There are currently no feed additives which will result in complete detoxification of vomitoxin contaminated feed.

Marketing Implications of Fusarium-Affected Grains

Don Bonner[1]
Canadian Wheat Board

CWB Role

What is our role at the CWB in marketing fusarium-affected grain? I see it as at least two-fold. First, as the sole marketer for export wheat and barley on behalf of Western Canadian farmers, we have an obligation to help farmers market their fusarium-affected grains. We are marketing for the long-term so we also need to meet the customer's needs and wants with the product we supply. As the farmer's marketing partner we need to make decisions that will make all farmers the most money without over-penalizing farmers with fusarium grain, or reducing overall quality and value of our grain.

Marketing to over 70 countries we have found that there is a wide range of acceptance or tolerance of fusarium-affected grain amongst our customers. Acceptance ranges from zero to no defined limit by country. What I mean here, is that for some countries there are no set government standards for imports into that country; it is up to the individual buyer to set the specifications if he or she wants to. These limits may depend on what the bulk product is being used for. Again, many governments may set limits for fusarium or DON levels for imported grains, but buyers often require a tighter specification than this. Even buyers not using the wheat for flour may have low tolerances. For example, 1 ppm DON wheat may be detrimental to the health of shrimp, even though it is generally considered safe for human consumption.

Customer Tolerances

Many Asian countries do not have government-set or even buyer-specified fusarium limits, but most Asian buyers are very quality conscious and expect their wheat to have good milling performance and reliable baking characteristics. Japan is one example in Asia, where there is essentially a zero DON tolerance, and it happens to be one of our best customers. It is very important for our farmers that we take steps to ensure that we are able to supply them, DON-free wheat.

Although, tolerance levels vary within Europe, one can categorize European and Middle-Eastern customers as having generally tight standards. The requirements in Latin America are more variable with some customers having higher tolerances of up to 4 ppm DON.

Farmer's Marketing Issues

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Looking at the marketing issues important to farmers I think the top would be that he or she wants to receive the maximum value for their grain. This is both a short-term and long-term issue. You may be able to move higher fusarium grain into certain markets in a given year and maximize short-term returns, but if this results in customer dissatisfaction with the product and a loss of market share in the future, this could reduce returns in the long-term for producers. Secondly, farmers want to be fairly compensated for their product, so we need to ensure that we are reflecting market value to farmers with fusarium grain.

History

The first major incidence of fusarium head blight in Western Canada occurred in 1993. At that time there were tight tolerances on #1 and #2 CWRS, and that unfortunately, resulted in a lot of crop in the Red River Valley failing to make the criteria. Back then, the CWB, the grain handling industry and farmers themselves, had little experience with fusarium and there wasn't a lot of information available on how to best handle and market the crop. In hindsight, a conservative approach was taken mainly because of the possible impact on our premium markets. A much greater understanding was attained during that first year and for the 1994/95 crop year, tolerances were increased to 2% fusarium-damaged kernels and a "High Tombstone allowance" program was put in place. The 2% level was set at that time using safety considerations.

The High Tombstone (later renamed to High fusarium) programs allowed producers to deliver higher fusarium grain and still receive milling quality values rather than be discounted to feed levels. They also compensated the handling company for blending and/or cleaning costs. Various handling agreements were put in place with the industry to segregate and/or blend fusarium and fusarium-free grains. These programs impact the efficiency of the handling system, require additional management of stocks and are therefore a cost to the farmer, but were nonetheless necessary to manage the problem of fusarium grains in our system.

In 1997/98 in particular we had many complaints from buyers and end-users regarding functionality problems with high protein 2 CWRS. In some cases loaf volume was as good with 12.5% protein CWRS as it was with 14.5% protein. This problem was treated very seriously, and our technical team, which included the Canadian Grain Commission, followed up on the problem with customers. From the initial investigation it was speculated that fusarium-damaged kernels may be the causing the problem, but it needed more research to back up that hypothesis. The CGC GRL concluded from their testing, that fusarium was a significant factor in the reduced performance. It was also concluded that midge-damaged kernels were also problematic for milling and baking properties.

As a result of the findings, tolerances were lowered on 2 CWRS and 1 and 2 CWES from 2% to 1% fusarium-damaged kernels and midge damage tolerances were also reduced. CWES is sold with the expectation for high gluten strength, and with weak gluten a major problem caused by fusarium, the tolerances needed to be lowered for all CWES grades.

Another important conclusion from the testing was that a sample of high fusarium wheat, say 8%, blended down to 2%, did not have the same good milling and baking characteristics at that of a sample that initially just had 2% damage. This is why we reduced the maximum tolerance on the high fusarium program from 10% to 5%. We feel that these two responses will re-establish the high quality reputation of our 2 CWRS grade. So far this year, fusarium levels in Thunder Bay unloads have been trending lower, indicating that either the fusarium problem was not as great this year, and/or the programs are working.

Another important factor not to be overlooked, is that everyone has learned how to deal with the problem better, starting with farmers, who are better able to incorporate innovative agronomic techniques and also adjust their combines to minimize sample contamination, to handling companies who are better able to clean, segregate and blend the bulk grain, to us, who are better able to deal with marketing issues.

Malting Barley

The problem with fusarium in malting barley was initially a boon for Canadian 6 row malting barley producers. The U.S. is a large and generally discriminating buyer of 6 row malting barley, and as their crop became largely affected by fusarium, it opened up a large market for 6 Row barley for us of up to half a million tonnes a year.
Because of the problems associated with fusarium in malting barley (for example: beer gushing), there have been good premiums ranging from about USD 0.30 to 1.00/bu for zero-DON malting barley in recent years.

Unfortunately, however, fusarium has become widespread across a large portion of our traditional 6 row malting region to the point where little zero DON barley is selectable from Manitoba. Most of our exports to the U.S. are now originating from northeastern Saskatchewan.

As a result of the decreasing supplies of good zero-DON barley in the U.S., U.S. maltsters have been using more and/or higher levels of DON-affected barley. How they are managing to do this, is likely a highly guarded trade secret, but they are doing it, and I assume, with some success.

If the fusarium problem continues to expand in the eastern Prairies it may cause a shift by both maltsters and farmers from 6 row to 2 row, which is less susceptible to fusarium.
Finally, and this is a comment to everyone in our industry from farmers to researchers, we have to keep our problem under control and need to continue to support various facets of research to maintain our quality and remain competitive. If fungicides or tolerant varieties are developed in the U.S. before they are available for our farmers we will lose our competitive edge.

Competitors

What are our major market competitors doing? The U.S. has no set export standards that I could find for fusarium per se, but it is considered part of the total kernel damage count. However, as many of their customers are the same as ours, stocks must be blended or segregated and monitored on loading to ensure that the shipment meets the needs and specifications of the customer. The onus therefore falls on each seller to carry this out.

Domestically, the U.S. Food and Drug Administration has implemented a maximum 2 ppm vomitoxin limit on flour. As a result, U.S. mills are not willing to accept wheat with more than 2% vomitoxin. It has been reported that U.S. elevators usually refuse grain with vomitoxin greater than 2 ppm and certainly heavily discount higher DON grain.
In Australia, the export standard is 2 ppm, but they don't really have a problem with fusarium, so I doubt that this level has ever been reached.

Future Actions

What we need to do in the interim? We need to continue to monitor the situation and put programs in place or revisit tolerance levels to best balance customer needs and concerns with producer returns. This includes: 1) ensuring that the new tolerances are working by following up with customer satisfaction queries; 2) monitoring the spread of the disease and its impact each year (this is something that our Weather and Crop Surveillance Department does), 3) monitor the impact of fusarium on other classes, which may become apparent if the disease spreads further west (this could result in changes to tolerance levels for other classes) and 4) reviewing handling programs to best manage the problem in each year's crop.

Conclusion

Because it is toxic, and also has a negative influence on flour processing and baking properties, fusarium will always be a serious marketing issue until resistant wheat and barley varieties with acceptable agronomic performance and quality are grown in susceptible areas, or until fungicides are able to provide complete control. Fusarium has caused marketing problems for us and increases our handling costs. The functionality problems fusarium causes will remain a threat to our reputation as a reliable supplier of quality grain and may even make it difficult to compete in certain markets.

Although everyone has learned a lot, and the system is much better able to cope with the problem, we have seen the disease spread geographically since it first struck in a major way in 1993. The disease has cost farmers millions of dollars in lost revenue. In order to maintain the viability and competitiveness of our farmers, the whole industry must continue to work together to solve this devastating disease. Forums such as this are part of that effort and we are happy to be part of it.

Breeding for Fusarium Head Blight Resistance in Wheat in Canada

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Spring Wheat

Soon after the first reports of severe Fusarium Head Blight (FHB) in southern Manitoba in the 1980's the Cereal Research Centre of Agriulture and Agri-Food Canada in Winnipeg initiated a program to breed for resistant wheat cultivars. The initial emphasis was on Canada Prairie Spring (CPS) and durum wheat as these classes appeared to be most severely damaged. Subsequent epidemics soon revealed that this disease was a serious threat to all the spring wheat classes grown in Manitoba. Clearly FHB has been moving westward and is a potential threat in a large part of the western Canadian prairie area. Breeding for FHB resistance is now a major objective of all the CRC wheat breeding programs and has recently become an import goal of breeding programs in Saskatchewan as well. The work at CRC will be discussed in this paper.

The major sources of resistance used for the hexaploid wheat classes are the varieties Suzhoe, Ning 8331, Sumai #3 and Nanjing 7840 which all originate from China. The initial crosses made at CRC were made with CPS wheats and the best lines selected from these crosses have been used extensively as parents in subsequent crosses for all classes of spring wheat. Derived lines commonly used as parents include FHB#20, FHB#21 and FHB#37. Similarly in bread wheat lines selected from initial crosses to the Chinese sources of resistance (RL 4802, 93B452-V2A and 93B42-AS1B) have now been used as the resistance source in our current crosses.

Because we were unable to identify any durum wheat lines with good levels of resistance to FHB the initial strategy in breeding FHB resistance durum wheat was to attempt to transfer the resistance from hexaploid wheat. This has not been successful. We have now identified lines of T. dicoccoides with good levels of resistance which are the basis of the current breeding programs.

The success of our breeding programs depends upon the screening methodologies developed and implemented by plant pathologists. In the early years of our program all the screening was done in the growth cabinets using a combination of point inoculation and spray inoculation. As the populations increased we turned to field nurseries which were inoculated by spraying at anthesis and again a few days post anthesis. The humidity was maintained at a very high level by using misting irrigation. In 1999 we further expanded our screening capability by establishing a second nursery utilizing Fusarium infected corn as the inoculum source. In order to maintain high humidity the whole 4 ac. nursery was given a light (0.1") irrigation every second or third evening from the time the inoculum was spread in the nursery until 4weeks after anthesis.

The current protocol for breeding FHB resistant wheats consists of: Screening F2

populations in the corn inoculated nursery that is also inoculated with leaf and stem rust; F3 is grown in New Zealand; F4 head rows are screened in the corn inoculated nursery that is also inoculated with leaf and stem rust; F5 is grown in New Zealand; F6 is grown in multi-location yield trials and also screened in the spray inoculated Fusarium nursery; F7 is grown in New Zealand; F8 is grown in multi-location yield trials and also screened in the spray inoculated Fusarium nursery; from there the lines are entered into the regional/national testing system of A, B and C level tests that are evaluated for FHB in replicated trails in the spray inoculated nursery.

The development of PCR markers has improved the efficiency of backcross breeding strategies. This technology allows us to select those single BCF1 plants with the markers for all 3 genes for screening in F2, while eliminating the progenies of the other F1 plants. Double haploidy is a second new technology to increase the efficiency of breeding for FHB resistance. The completely homozygous and homogeneous nature of the progeny of doubled haploid plants makes the characterization of the FHB reaction much easier than in conventional segregating generations. In addition double haploid results in higher frequencies of plants that carry all the desirable genes in the homozygous state.

The status of the CRC programs in 1999 was: CWRS - 2 lines in the 1st year Coop, 3 lines in B test, 37 lines in A3 test and 286 F8 or DH lines: CPS - 1 line in 2nd year Coop, 11 lines in B test, 62 lines in A test, 107 F8 lines and ½ to 3/4 of early generation lines; CW Extra Strong - 2 lines in B test, 12 lines in A test, 6 F4 populations and 2 F2 populations: and in Durum - several BC1F2's and BC3F2's populations.

Winter Wheat

The Eastern Cereal and Oilseed Research Centre of Agriculture and Agri-Food Canada in Ottawa has had an ongoing program to breed for FHB resistance for many years. Initial crosses were made with the soft white winter wheat cultivars, Harus, Frederick and Augusta with a Brazilian spring wheat cultivar Frontana to transfer resistance. Genetic stocks, developed from these crosses were evaluated at ECORC. A number of lines were identified as resistant/tolerant. The goal is to develop variety as resistant to FHB as the best of the first cycle selections, that would be commercially competitive in yield, quality and adaptation. At ECORC FHB tolerance from a donor spring type parent, Frontana was successfully transferred into winter wheat. Four winter lines (FHB 143, 147, 148 and 161) have been used in crosses with commercial cultivars in order to recover further yield and quality parameters. In addition, the Chinese lines Sumai 3, Wongshubai, Wuhan and the Ning series: the European lines Ringo Star, WW Capo, Renan, SVP 72017, and U136 lines: and the Russian lines, Dakha, and Soratnitso have been recently used as sources of resistance.

At ECORC the segregating generations are screened in a mist irrigated nursery utilizing spray inoculation at anthesis and 7 days later. Visual symptoms are rated and DON values are determined (by ELISA techniques). During 1998-99 crop year 130 lines

selected in the F5 generation in 1997-98 were evaluated and the top 20% of the lines based on agronomy, yield and FHB tolerance were selected for further evaluation. In a collaborative project with W.G. Thompson and Sons Limited over 3000 pure breeding lines were developed from the crosses involving commercial cultivars and the four winter type FHB resistant parents (FHB 143, 147, 161 and 148) developed at ECORC. In 1998-99 541 double haploids were evaluated at Ottawa and Nairn, Ontario. Selected lines (with FHB tolerance) are currently at the seed increase stage for a multi-location yield trials. And these lines are also plated in the FHB nursery for second cycle of inoculation and rating.

Breeding for Fusarium Head Blight Resistance in Barley.

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Introduction

Most barley breeding programs in Canada have only recently started to breed for resistance to Fusarium head blight (FHB). Efforts in the United States have been underway longer as a result of the severe FHB epidemics in Minnesota, North Dakota and South Dakota from 1993-98. In eastern Canada, breeding for FHB resistance began in the mid-1990's at the Eastern Cereal Research Centre (ECORC), Ottawa, followed more recently by private breeding programs in Quebec. In western Canada, active breeding efforts began with the spread of FHB beyond the Red River Valley into western Manitoba and eastern Saskatchewan. We made our first crosses at Brandon in 1996. All breeding programs in western Canada are now involved.

Although FHB can cause significant yield losses, quality and market acceptability issues are more critical in barley. The American malting and brewing industry has been testing all barley samples for deoxynivalenol (DON) mycotoxin produced primarily by *Fusarium graminearum*. DON must usually be "non-detectable" (< 0.5 ppm) to be accepted by the industry. This will be a very difficult target to hit in breeding for FHB resistance in malting barley. The brewers are concerned about gushing problems related to FHB, but perhaps more importantly, public perception problems if mycotoxins are found in beer. Domestic feed plants are also testing for DON and they may refuse barley, particularly for swine rations, if DON is detected.

The objectives of my presentation then will be to: 1) examine the FHB resistance of our current barley varieties, 2) discuss problems in improving FHB resistance in barley, and 3) outline breeding strategies for improving FHB resistance in barley.

FHB Resistance of Current Barley Varieties

Since DON concentration is the ultimate measure of FHB resistance, I will now present some data collected by Andy Tekauz and Brent McCallum of the Cereal Research Centre, Winnipeg, from variety trials grown under natural or artificially inoculated conditions over a range of environments in Manitoba in 1997 and 1998 (Table 1). Because the varieties differed from test to test, AC Metcalfe was used as the check with which to compare each variety since it was the only one grown in all tests. AC Metcalfe is a two-row malting barley variety with moderate resistance to FHB. Note that the number of tests is given for each variety comparison. The letter after the number of tests indicates that a different set tests were used to calculate the mean although there may be some tests in common. Varieties with only one or two sites were probably grown in 1998 when infection levels were generally lower so must be interpreted with caution.

The two-row malting varieties appear similar to AC Metcalfe as a group and have the best resistance to FHB compared with the other classes of barley (Table 1). However, these

varieties will still be affected by FHB and accumulate enough DON to be rejected

Table 1. Deoxynivalenol (DON) concentration (ppm) of barley varieties grown in Manitoba in 1997 and 1998

Variety	DON	AC Metcalfe[1]	N[2]	Variety	DON	AC Metcalfe[1]	N[2]
Two-row Malting:				***Six-row Malting:***			
AC Bountiful	0.4	1.2	2a	B1602	1.1	0.8	1b
AC Oxbow	1.2	2.4	4a	BT950	1.2	0.8	1b
CDC Kendall	1.5	0.8	1b	CDC Sisler	0.9	1.0	3a
CDC Stratus	0.5	1.0	3a	Excel	2.6	1.4	2b
Harrington	1.3	1.0	3a	Foster	3.0	1.0	3a
Manley	3.0	2.9	3b	Robust	2.5	1.0	3a
Merit	0.7	1.2	2a	Stander	6.4	2.3	5a
				Argyle	3.7	2.2	5b
				Tankard	3.7	2.9	3b
Two-row Hulless:				***Six-row Hulless:***			
CDC Dawn	1.1	0.6	2c	AC Bacon	0.9	0.6	2c
CDC Freedom	0.5	0.3	1a	AC Hawkeye	0.7	0.3	1a
CDC Gainer	0.8	0.6	2c	CDC Silky	2.5	2.0	5c
Condor	2.8	0.8	1b	Falcon	1.5	2.4	4a
Two-row Feed:				***Six-row Feed:***			
AC Sterling	1.2	0.8	1b	AC Harper	1.9	1.2	2a
Bridge	3.9	2.9	3b	AC Lacombe	5.8	2.4	4a
CDC Fleet	7.5	1.2	2a	AC Rosser	6.3	1.7	6a
CDC Guardian	1.7	2.9	3b	Bedford	5.1	2.7	4b
				Bronco	1.8	1.2	2a
				CDC Earl	6.7	2.4	4a
				Duke	5.3	2.9	3b
				Gamine	1.7	1.2	2a
				Heartland	9.3	2.9	3b

[1] Mean DON concentration (ppm) of AC Metcalfe for same tests as the variety.
[2] Number of tests used to calculate the mean, where numbers followed by the same letter represent the same set of tests.

by the malting and brewing industry if conditions are right for the pathogen. We still need to improve on this level of resistance. Harrington and Manley may be somewhat more susceptible since they usually have a higher FHB severity rating. Visual ratings for FHB in barley can be confused with symptoms of black point which is usually incited by *Cochliobolus sativus*. Since Harrington and Manley are highly susceptible, it is possible that infection by *C. sativus* may reduce FHB and DON concentration in these varieties.

As expected, the six-row malting varieties accumulated more DON than AC Metcalfe, particularly Stander (Table 1). An exception was CDC Sisler from the University of Saskatchewan, which was similar to AC Metcalfe over three tests. More testing will be needed to confirm CDC Sisler's FHB resistance, but it does look promising for a six-row white-aleurone malting variety. Tankard, a sister line of CDC Sisler, also looks promising in terms of DON concentration, but it has a blue aleurone and hence limited market potential.

Because DON is concentrated in the outer layers of the seed, hulless varieties would be expected to have less DON since the hulls are left in the field at harvest. This would seem to be the case since the DON concentration of both two-row and six-row hulless varieties generally approach that of AC Metcalfe (Table 1). However, Condor appeared to accumulate significantly more DON than AC Metcalfe in the one test in which it was evaluated. Although Falcon had a lower DON content than AC Metcalfe, Falcon has consistently had higher FHB visual ratings than the other varieties in this group and may be more susceptible to *C. sativus*.

In the two-row feed group, AC Sterling approached AC Metcalfe in DON concentration (Table1). AC Sterling is an eastern Canadian feed barley variety released by ECORC that has shown moderate resistance to FHB. Morrison and Symko, both from ECORC, have also performed well in other tests (data not shown). All three have Rodeo in their pedigrees. CDC Fleet was the most susceptible variety in this group. Although CDC Guardian appears to be the most resistant based on its DON concentration, it is extremely susceptible to *C. sativus*.

The six-row feed group accumulated significantly more DON than AC Metcalfe as expected (Table 1). Heartland, CDC Earl, AC Rosser and AC Lacombe were particularly susceptible.

Problems in Improving FHB Resistance of Barley

The most serious problem in improving the FHB resistance of barley is that the sources of resistance identified to date are not very good. One of the best sources of resistance is the Chinese accession, CI 4196 (Steffenson, 1999). It is more resistant to FHB than our two-row malting varieties, but it will still accumulate DON at levels greater than 0.5 ppm under heavy pressure from the pathogen. Most of the sources of resistance, including CI 4196, are two-rowed. The following two-row sources of resistance have been used in breeding programs: Svanhals, Zhedar 1, Zhedar 2, Zaoshu 3, Harbin, Germany II, Frederickson, Seijo II, Kyoto Nakate, Horni Peseky 2, Imperial, CI 8826, Gobernadora,

Atahualpa, Shyri, Misc Calidad 21, Gob/Humai10, Arupo/K8755//Mora, and others. Chevron is the best six-row source of resistance identified to date, but its level of resistance is not as good as that of the two-row accessions listed above (Steffenson 1999). Very few other six-row resistance sources have been identified, although the ECORC breeding program has been using CIMMYT-6. Attempts to transfer the FHB resistance from two-row accessions to elite six-row germplasm have generally met with failure, although crosses involving CI 4196 have shown promise (R. Horsley, personal communication). Most of the resistance sources are from northern Europe, China, Japan and Mexico (CIMMYT). They tend to be tall, late, weak strawed, low yielding and poorly adapted, as well as susceptible to such diseases as net blotch, spot blotch and stem rust. Incorporating these sources of resistance into elite breeding material will be a long term project.

The quantitative inheritance pattern of FHB resistance in barley is another major difficulty to overcome in developing new FHB resistant varieties. In a recent study using 101 $F_{4:7}$ lines from the Chevron/M69 cross, de la Pena et al. (1999) identified 10 quantitative trait loci (QTLs) for FHB resistance (% severity), 11 QTLs for kernel discoloration (black point), and 4 QTLs for DON accumulation. The majority of the QTLs conferring resistance as determined by these three traits came from Chevron. They also found 7 QTLs for heading date and 6 QTLs for height, with most of the QTLs for tall, late plants also coming from Chevron. A number of these QTLs were in the same chromosomal region as the QTLs for one or more of the measures of FHB resistance. It could not be determined whether this association was due to linkage or pleiotropy. However, 5 regions associated with at least one of the three measures of resistance but not with heading or height were identified as having potential for molecular marker assisted selection (MAS).

MNBrite, a six-row malting barley variety recently released by the University of Minnesota (Rasmusson et al. 1999), recovered some but not all of the FHB resistance of Chevron after 8 cycles of breeding and selection for kernel discoloration. It is more resistant to FHB than varieties such as Robust, but not as resistant as Chevron and can be overwhelmed by the pathogen under moderate to heavy infection levels (Steffenson 1998). Clearly, more resistant varieties are needed, but varieties like MNBrite may be useful in the short term.

FHB resistance appears to be confounded by relationships among genetic, physiological and morphological traits. As noted previously, the sources of FHB resistance tend to be tall and late. What is the biological basis of FHB resistance anyway? Are FHB resistant accessions truly resistant or is resistance associated with morphological or physiological traits, such heading date, height and inflorescence traits? In a recent study, Zhu et al. (1999) found that the principal QTLs for FHB resistance in a doubled haploid (DH) population from the two-row cross Gobernadora/CMB643 were coincident with QTLs for inflorescence traits and height. Both parents were from the CIMMYT program. Understanding these relationships will be key in developing new FHB resistant varieties.

One of the problems in working with FHB in barley is that the visual symptoms of FHB are not distinct and can be easily confused with other diseases such as black point which is incited mainly by *C. sativus*. Visual ratings are not very reliable so we are more dependent on measuring DON concentration in barley. However, we can not evaluate everything for DON concentration because of cost. It is also desirable to discard breeding lines that are particularly susceptible to *C. sativus* and produce "FHB-like" symptoms.

Barley appears to have a wider window of infection after anthesis than wheat. It is possible for FHB to come in later with few visual symptoms and still increase DON concentration. Thus, resistance must protect barley for a longer period of time.

Pathogen variability is a potential problem. Many different isolates of *F. graminearum* have been identified in the same field so we know that the pathogen is highly variable. Will we be able to keep ahead of it with genetic resistance? Also, at least four other species of *Fusarium* may cause FHB in barley (Salas et al. 1999). Since *F. graminearum* appears to predominate once it moves into an area, we are justified in concentrating our screening efforts on it and measuring DON concentration. However, we must keep the other *Fusarium* species in mind and try to use resistance that is as broad-based as possible.

FHB screening nurseries are time-consuming, labour intensive and expensive. This is, of course, not unique to barley. The nurseries can be difficult to establish as infection levels may be too low or too high to give good results. Another concern is the lack of repeatability in FHB nurseries. A line must be tested many times before we can be confident of its FHB resistance.

As noted previously, testing for DON accumulation is very expensive but of paramount importance in barley.

A final impediment is the need to improve other traits as well. As breeders, we must deal with other diseases, quality parameters and agronomic goals that are constantly changing. We must move forward on the whole package and not just focus on FHB resistance if we are to develop new varieties with economic value.

Breeding Strategies for Improving FHB Resistance

Winter screening nurseries in China are being used by a number of programs in the United States and by ECORC. FHB is the only disease in these nurseries, and the symptoms are much more distinct. There has been fairly good agreement with North Dakota results. Use of Chinese winter nurseries can greatly hasten advancement and assessment of breeding lines with FHB resistance.

Expanding screening efforts in FHB nurseries is effective but takes time, resources and money. FHB nurseries are still the best way to assess natural accumulation of DON in barley accessions. North Dakota State University (NDSU) evaluated nearly 30,000 breeding lines in 1999 (Steffenson 1999). In Canada, we need to adequately screen our

advanced breeding lines as well as segregating material from crosses designed specifically for FHB resistance. We must expand our capacity in western Canada which was only about 5,000 barley lines (Brandon and Glenlea combined) in 1999. This is no where near the capacity that we need.

Molecular mapping of FHB resistance will be useful to breeders. The knowledge concerning chromosomal location of resistance loci may facilitate efficient selection and transfer of desired traits. We will have much more information in a year or two when the results of several studies on the molecular mapping of FHB resistance in such accessions as CI 4196 and Zhedar 2 are published. These projects will also help to locate coincident QTLs for resistance and undesirable traits. Most of this work is being conducted in the United States, with some at ECORC as well. There are many advantages to MAS, but it may be limited in practice by cost, large number of genes, large populations, and tendency for markers to be cross specific.

Male sterile facilitated recurrent selection is being used at NDSU to accumulate FHB resistance genes and other desired traits.

Use of doubled haploids may accelerate the development of FHB resistant breeding lines and parents. It may be especially beneficial when used in combination with MAS.

In vitro selection for resistance to FHB in tissue culture is another method that could be used. We have initiated a project at Brandon to use DON or other mycotoxins as selection agents in our anther/microspore culture doubled haploid system. We hope to increase the FHB resistance of regenerated lines. Some success has been reported in wheat.

Genetic transformation is perhaps the most exciting possibility for developing varieties with a high level of FHB resistance. Transformation protocols have been developed in barley and are being fine tuned to work with elite germplasm at local facilities. Research is in progress at Washington State University to engineer the blockage of the dihydroflavanol reductase gene in the seed in order to accumulate an anti-fungal compound (Steffenson 1998). Researchers at the University of Minnesota are attempting to utilize anti-fungal proteins from barley and other species. Some success has been reported in wheat. Research is also in progress at the Cereal Research Centre, Winnipeg, to use genetic transformation to improve FHB resistance in barley.

Conclusions

Despite the difficulties, progress is being made in the United States in improving the FHB resistance of barley. This is encouraging for those of us who have started working on this problem in Canada. It will probably take a long while though to develop highly resistant varieties. Genetic transformation looks particularly promising as a technique for improving the level of FHB resistance in barley. However, genetics alone will probably not solve the FHB problem, but should be used in combination with other control methods.

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References

de la Pena, R.C., Smith, K.P., Capettini, F., Muehlbauer, G.J., Gallo-Meagher, M., Dill-Macky, R., Somers, D.A., and Rasmusson, D.C. 1999. Quantitative trait loci associated with resistance to Fusarium head blight and kernel discoloration in barley. Theor. Appl. Genet. 99:561-569.

Rasmusson, D.C., Wilcoxson, R.D., Dill-Macky, R., Schiefelbein, E.L., and Wiersma, J.V. 1999. Registration of 'MNBrite' barley. Crop Sci. 39:290.

Salas, B, Steffenson, B.J., Casper, H.H., Tacke, B., Prom, L.K., Fetch, T.G., Jr., and Schwarz, P.B. 1999. *Fusarium* species pathogenic to barley and their associated mycotoxins. Plant Dis. 83:667-674.

Steffenson, B.J. 1998. Fusarium head blight of barley: epidemics, impact, and breeding for resistance. Tech Quart. MBAA 35:177-184.

Steffenson, B.J. 1999. Combating *Fusarium* head blight: an emerging threat to malting barley quality throughout the world. Pages 531-539. *In* EBC Congress 1999.

Zhu, H., Gilchrist, L., Hayes, P., Kleinhofs, A., Kudrna, D., Liu, Z., Prom, L., Steffenson, B., Toojinda, T., and Vivar, H. 1999. Does function follow form? Principal QTLs for *Fusarium* head blight (FHB) resistance are coincident with QTLs for inflorescence traits and plant height in a doubled-haploid population of barley. Theor. Appl. Genet. 99:1221-1232.

Breeding for Resistance to Ear Rot in Corn

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Since 1986, AAFC has been breeding for increased resistance to ear rots in corn as part of a multiple-pest resistance breeding program. Three *Fusarium* species are predominantly responsible for this disease: *F. graminearum*, *F. moniliforme*, and *F. subglutinans* (Vigier et al., 1997). The mycotoxins of most concern are those produced by *F. graminearum* (deoxynivalenol and zearalenone) and *F. moniliforme* (fumonisins). Only a few commercial hybrids possess moderate resistance to these pathogens; however, these hybrids tend to be lower yielding. *Fusarium* species enter the corn ear through two major modes: (1) by growth of mycelium down the silks to the kernels and cob (rachis) from spores germinating on the silks; and (2), by entry through kernel wounds created by insects or birds. Silk infection is believed to be the major mode of entry during epidemics; however, breeding for resistance must be aimed at both modes since there is no association between resistance to kernel and resistance to silk infection.

Since the sporadic nature of ear rot epidemics in Canada made natural infection unreliable, one of the first challenges in developing an ear rot resistance breeding program was the development of screening techniques to evaluate genotypic resistance. When the program started in 1986, techniques were unavailable and there were no known sources of resistance. Since that time, we have developed inoculation techniques for both modes of fungal entry. For 5 years, these techniques were extensively studied to evaluate the many parameters associated with their use, with the aim of standardizing the techniques for routine use in a breeding program which screens thousands of plants per year. Parameters studied included: inoculum production protocols, time of inoculation, spore concentration, inoculum volume, position of inoculation, isolate effects, irrigation requirements, association to mycotoxin levels, and evaluation of disease symptoms (Reid et al., 1996a). Both inoculation techniques allow for good differentiation between genotypes, ranging from very susceptible to highly resistant.

The silk technique consists of injecting a spore suspension into the silk channel inside the husk cavity and above the cob. In a breeding nursery, pollinations are conducted as usual, then ear shoot bags are lifted to perform inoculations and replaced. Two ml of inoculum are injected into the silk channel of each primary ear using a syringe or other apparatus. The rate of progression of the fungus down the silk channel is a function of the degree of inherent silk resistance, silk age and the environment. Inoculations must be conducted 4-7 days after silk emergence since silks rapidly senesce after pollination and this physiological change alters the suitability for growth of ear-rotting organisms. Inoculations made later than 8-10 days post-silk emergence will result in very little infection.

The kernel inoculation technique involves wounding the husk and kernels by stabbing

them with four small (3 mm dia.) stainless steel pins previously dipped in spore suspension or by injecting inoculum into the kernels. The technique wounds 3-4 kernels thus producing a point source of infection from which the fungus spreads. Infection usually spreads around the circumference of the ear first and then moves to the tip rather than the butt of the ear since the butt kernels are dryer; the degree of spread is highly dependent on genotypic resistance and the environment. Timing of inoculation is not as critical as with silk inoculations but best results are achieved if inoculations are conducted 10-15 days after silk emergence (blister to early milk stage).

For most hybrids and inbreds, it takes approximately 6-8 weeks for symptoms and toxins to reach a peak and stabilize. After this time, ears are hand husked and the severity of symptoms is evaluated using a rating scale based on the percentage of kernels with visible symptoms of infection such as rot and mycelial growth. When making selections in a breeding nursery, the acceptable level of visual symptoms is dependent on the technique used. For silk inoculations, ears with no visible infection reflect resistance to the spread of infection down the silk channel. With kernel inoculations, there is always some symptoms; resistant plants are those in which the infection does not spread from the wounded to the non-wounded kernels, except in extreme cases where the wounded kernels abort.

A strong positive correlation ($r>0.80$) exists between visible disease symptoms and toxin levels, thus it is not necessary to conduct mycotoxin analyses at all stages of inbred development (Reid et al., 1996b). Selection can be based on visual evaluation of disease symptoms. Toxin analyses are desirable in the final stages of inbred and/or hybrid development before release because the acceptable level of toxin in feed is low.

Using these techniques, genotypes with resistance to one or the other mode of infection have been identified in adapted germplasm and insights into the inheritance of resistance to each mode of entry have been made. Both silk and kernel resistance is highly dominant; silk resistance appears to be qualitative (Reid et al., 1994), while kernel resistance is largely quantitative with strong additive effects (Chungu et al. 1996).

One of the first identified sources of resistance is the AAFC inbred CO272. This inbred possesses moderate silk resistance but no kernel resistance. Unfortunately, its agronomic performance is very poor. Another AAFC inbred, CO325, was found to possess moderate kernel resistance. In the development of new lines, these inbreds and others were used as donor parents followed by inbreeding, inoculation and resistance screening for each of several generations. At harvest, only those ears with no visible symptoms of infection on the kernels were selected and advanced to the next generation. In experimental test crosses with susceptible checks, hybrids had outstanding resistance and yield when artificially infected via the silk or kernel, depending on the original source of resistance. In addition to breeding from inbred sources, we have collected germplasm from around the world (adapted and unadapted) with moderate to high resistance to various ear pathogens. This material has been screened for *Fusarium* resistance and those showing promise have been incorporated into the breeding program. In 1988, six

commercial hybrids were identified with intermediate resistance and used to initiate the development of a 'fusarium resistant synthetic' from which several inbred lines are being developed.

AAFC has released 5 inbreds with improved resistance to ear rot. The first three (CO387, CO388 and CO389), released in 1996, possess good silk resistance but poor kernel resistance. The source of resistance in these lines was CO272. In 1999, the first two inbreds (CO430 and CO431) developed from the fusarium resistant synthetic were released. All five inbreds are flints. CO387, CO430 and CO431 are early inbreds. CO388 and CO389 are late in maturity since B73 is in their pedigree. So far, CO388 has exhibited the best combining ability of all five. In the summer of 1999, we identified a medium maturity dent inbred with very high resistance developed from the synthetic; this inbred will be released in 2000. As well, four other flint inbreds of a different pedigree may be released. All have been selected for resistance as well as agronomic performance so that yield losses will be nil or minimal when the inbred is used in a hybrid combination. Several seed corn companies are currently developing hybrids with these inbreds; it is expected that the first hybrid with improved resistance to ear rot will be commercially released within 1-2 years.

References

Chungu, C., Mather, D.E., Reid, L.M. and Hamilton, R.I. 1996. Inheritance of kernel resistance to *Fusarium graminearum* in maize. J. of Heredity 87: 382-385.

Reid, L.M., Hamilton, R.I., and Mather, D.E. 1996a. Screening Maize for Resistance to Gibberella Ear Rot. Agriculture and Agri-Food Canada Technical Bulletin 1996-5E.

Reid, L.M., Mather, D.E., Bolton, A.T. and Hamilton, R.I. 1994. Evidence of a gene for silk resistance to *Fusarium graminearum* Schw. ear rot of maize. J. of Heredity 85: 118-121.

Reid, L.M., Stewart, D.W., and Hamilton, R.I. 1996b. A 4-year study of the association between gibberella ear rot severity and deoxynivalenol concentration. J. Phytopathol. 144: 431-436.

Vigier, B., Reid, L.M. Seifert, K.A., Stewart, D.W., and Hamilton, R.I. 1997. Distribution and prediction of *Fusarium* species associated with maize ear rot in Ontario. Can. J. Plant Pathol. 19: 60-65.

Fusarium Head Blight in Oat

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Oat is often grown in regions where Fusarium head blight (FHB) affects wheat and barley. In North America it is not known if this disease is prevalent or damaging in oat but this has been investigated in Scandinavia (Weizhong *et al.* 1997, Langseth and Elen 1996, Lanseth and Stabbetorp 1996). The general conclusions from these studies were: (1) *Fusarium* spp. can be isolated from oat seeds although visual symptoms of FHB are not apparent, (2) the causal species is primarily *Fusarium culmorum*; (3) the levels of seed infestation and DON are correlated; and (4) lodging of the crop leads to a significant increase in DON content.

In 1998, grain of AC Barrie, AC Vista, and AC Intrepid wheat, AC Rosser and Argyle barley, and AC Assiniboia and Triple Crown oat were sampled at each Manitoba Crop Variety Evaluation Trial (MCVET) location. Twenty grams of harvested grain from 3 replicates were sub-sampled and ground, and 1 gm subsequently used for DON assay using ELISA (enzyme linked immuno-sorbant assay). Results from the 5 locations with the highest overall DON levels are listed in Table 1.

Table 1. DON (ppm) levels in AC Vista wheat, AC Rosser barley, and Triple Crown and AC Assiniboia oat from 1998 Manitoba Crop Variety Evaluation Trials (MCVET)

	Wheat	Barley	Oat	Oat	
Location	AC Vista	AC Rosser	Triple Crown	AC Assiniboia	Average
Rosebank	23.9	3.6	2.3	1.7	7.9
Hamiota	9.6	4.0	2.0	2.0	4.4
Brandon	2.4	1.1	3.6	2.5	2.4
Neepawa	6.0	2.5	2.2	1.8	3.1
Boissevain	2.8	1.2	1.5	1.4	1.7
Average	8.9	2.5	2.3	1.9	3.9

As levels of DON in oat were highest at the Brandon location, oat cultivars grown at that site were subsequently assayed for DON (Table 2). During industrial processing , oat hulls are removed resulting in 'groats'. These may be processed further, using heat and

high humidity, a treatment known as 'conditioning' or 'kilning'. Twenty gram sub-samples of each variety were mechanically dehulled and the resulting groats ground and assayed for DON. The groat samples from AC Assiniboia and Triple Crown were subsequently conditioned, then ground and assayed for DON. The groats contained approximately 50% of the DON in whole oat samples from the same plots. In conditioned oat, DON was at 0.1 ppm, which is at the detection level for the assay. DON may be present within conditioned oat but in a form not readily detectable using ELISA.

Table 2. DON (ppm) levels in whole oat, groats and conditioned oat grown at Brandon MB, 1998

Variety	Whole oat	Dehulled oat	Reduction (%)	Conditioned oat
AC Assiniboia	2.3	1.1	53	0.1
CDC Boyer	1.7	0.5	69	
Triple Crown	1.6	0.8	47	0.1
AC Rebel	1.3	0.7	45	
Gem	1.2	0.5	57	
	1.6	0.7	54	0.1

To determine the *Fusarium* spp. and their levels in oat, 100 seeds from each of 3 replicated plots of AC Assiniboia and Triple Crown from Brandon were surface sterilized and plated onto potato dextrose agar (PDA). Total *Fusarium* spp. infection averaged 8.2%, *F. graminearum* predominating at 4.5% but *F. avenaceum*, *F. sporotrichioides*, *F. equiseti*, *F. poae*, and *F. culmorum* were also recovered (Table 3).

Table 3. Levels of *Fusarium* in oat seed grown at Brandon MB, in 1998

		Fusarium spp.					
Variety[a]	Total	*graminearum*	*poae*	*sporotrichiodes*	*equiseti*	*avenaceum*	*culmorum*
AC Assiniboia	23	13	0	1	1	8	0
Triple Crown	26	14	1	3	2	5	1
Total	49	27	1	4	3	13	1
Percent	8.2%	4.5%	0.2%	0.7%	0.5%	2.2%	0.2%

[a] 300 seeds were plated for each variety.

In summary: (1) oat in Manitoba is infected by *Fusarium* spp., although no obvious visual symptoms of FHB are observed; (2) oats accumulate DON; (3) hull removal reduces

DON content by approximately 50%; (4) conditioning reduces DON to non-detectable levels (but it may still be present in a form undetectable by ELISA); and (5) there appear to be differences in oat cultivar reaction to FHB, suggesting breeding for resistance is possible.

References

L. Weizhong, W. Langseth, H. Skinnes, O. Elen and L. Sundheim. 1997. Comparison of visual head blight ratings, seed infection levels, and deoxynivalenol production for assessment of resistance in cereals inoculated with *Fusarium culmorum*. Eur. J. Pl. Pathol. 103:589-595.

W. Langseth, and O. Elen. 1996. Differences between barley, oats and wheat in the occurrence of deoxynivalenol and other trichothecenes in Norwegian grain. J. Phytopathology 144:113-118.

W. Lanseth and H. Stabbetorp. 1996. The effect of lodging and time of harvest on deoxynivalenol contamination in barley and oats. J. Phytopathology 144:241-245.

Chemical Control of FHB

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Introduction

Recent Fusarium head blight (FHB) epidemics in wheat in the United States and Canada have caused enormous yield and quality losses in both spring and winter regions (McMullen et al., 1997). Control of this disease has been difficult, because of the complex nature of the host/pathogen/environment interaction. Host resistance looks promising (Bai and Shaner, 1996; Stack, 1999), but full resistance in adapted cultivars is still sometime in the future, especially in certain winter and durum wheats. In the meantime, growers need immediate solutions for keeping this disease from causing economic losses.

Chemical control would be a promising immediate solution, if effective and affordable products were available. Results in Europe (Mauler-Machnik and Zahn, 1994; Mesterhazy and Bartok, 1996) indicated substantial control of this disease with a variety of products, many of which were not registered in the United States or Canada, or were not registered for full heading or flowering application, the time of infection of wheat by the scab fungus. Early work in the United States had limited or variable results. In Arkansas, Milus and Parsons (1994) used standard leaf disease control techniques to apply a number of different fungicides, and applied them at 50% heading. In these studies, fungicides did not significantly reduce scab or increase yield.

The severity of the recent FHB epidemics in North America necessitated a new look at finding safe fungicides and methods of applications that would improve chemical control. Traditional products and methods were not providing adequate results under severe epidemic conditions.

Problems

Product availability: For most of the 1990s in the United States, registered wheat fungicides effective against FHB were few and far between. Only two effective products were registered for heading application, benomyl (Benlate) and mancozeb (Dithane, Penncozeb, Manzate). Benomyl fungicide is a locally systemic product with good activity against Fusarium, but with lesser activity against the leaf spot fungi or rust fungi that also plague wheat. Benomyl is labeled for application to wheat in the US, up to 21 days before harvest. The mancozeb fungicides are protectant fungicides, not systemic, and they must be applied several times to wheat to persist long enough for adequate disease control. The mancozebs have good activity against leaf rust and leaf spot fungi, but lesser activity against Fusarium than benomyl. The mancozebs are labeled for application to wheat up to 26 days before harvest, which also generally allows application through flowering. Use of a combination of benomyl at 0.5 lb/acre and mancozeb at 1 lb/acre applied at flowering has given some protection against FHB, but it hasn't always been consistent in performance and does not control leaf diseases adequately, as well. In

our work in North Dakota, we found that the wettable powder formulations of these products often subjected workers to fungicide exposure and often plugged sprayer nozzles if not adequately agitated in the sprayer tank. The cost of the combination for one application was generally high for wheat, around $12/acre. Bravo or chlorothalonil fungicide also is a protectant fungicide, labeled for wheat in Canada, but not in the US. Propiconazole (Tilt) also is registered for wheat in the US, a locally systemic product with a wide spectrum of activity, but its full federal label allows only for application through early flag leaf emergence, a timing too soon for control of FHB. The following characteristics in a fungicide were needed: registered for heading application; economic; safe; systemic; easy to mix; and with a wide spectrum of activity against FHB and other important fungal wheat diseases.

Application methods: The second problem with chemical control of wheat FHB was that application methods used to control leaf diseases were being used to control head FHB. The Fusarium fungus infects the wheat head at flowering and continues to develop in the grain through soft dough stage. Fungicides must reach the target site of infection - the flowers and other grain head parts. Systemic fungicides, such as benomyl, are only locally systemic - they do not move upward from the leaf into the head. Thus, application to foliar parts of the wheat plant does not result in head FHB control.

Traditional methods of application of fungicides to wheat were designed to control leaf diseases by covering the flag leaf, which is generally flat or horizontal to the spray pattern. Benomyl and mancozebs were generally applied with a spreader/sticker in fairly large droplet sizes to reach the leaf target. The grain head presented a much different and more difficult target. It is vertical to the spray pattern, awns or beards may be present in many cultivars and they capture the spray before it reaches the site of infection, and the wheat glumes are glabrous or waxy, unlike the hairy leaves, and don't readily absorb the spray. Studies of various application methods were needed to determine if adaptations could be found that would increase fungicide coverage on the wheat heads, and hence improve FHB control.

Progress

Cooperative fungicide trials: In response to the recent major FHB epidemics in the United States, cooperative fungicide trials were established across many wheat classes and environments. Following the 1993 epidemic in the spring wheat region of North Dakota, Minnesota, and South Dakota, a number of trials were established to evaluate fungicide efficacy against FHB. Preliminary results with the registered products indicated disease control was best achieved if fungicides were applied to flowering wheat, not prior to or after flowering. Results from research studies led to some new special registrations of products for 1998.

A Section 18 Emergency Exemption for Folicur (tebuconazole) for heading application was granted by EPA to a number of states in 1998, and again in 1999. Many state labels (24C) were granted for Tilt (propiconazole) for use at heading time on wheat in 1998 and again in 1999. The availability of these two systemic products gave wheat growers

additional flexibility in applying fungicides to control FHB, the products cost about $9.00/acre, were easy to mix, did not plug nozzles, and lowered applicator exposure. In 1999, a full federal registration was granted to Quadris (azoxystrobin) for application to wheat up to 45 days prior to harvest.

Much of the progress made in getting additional fungicides available for FHB control was made possible through cooperative efforts across states to establish uniform fungicide trials. In 1998, seven states (IN, KY, MN, MO, ND, OH, SD) participated in this uniform fungicide trial that tested five fungicides or fungicide combinations against wheat FHB. In the three states with the most severe FHB, the average reduction in severity of FHB ranged from about 30 to 50%, and yield increases averaged up to 16%, but higher levels of control (up to 73%) and higher yields (up to 45%) were achieved in some trials. Levels of vomitoxin (DON) also were reduced from 28 to 56% and leaf diseases across all seven states were reduced by 37 to 69% (benomyl + mancozeb had least reduction in leaf disease). In 1999, the uniform fungicide trial was expanded to 14 states (AR, IL, IN, KY, MD, MI, MN, MO, NC, ND, NY, OH, SD, VA) and to nine fungicide treatments. Some states in 1999, such as OH, NY, and NC, had no wheat FHB because of severe drought, but a summary from other states with FHB indicated that Folicur , as well as Stratego (a combination of Flint and Tilt) and BAS 500 (a strobilurin) performed well against this disease (McMullen, M., et al. 1999). A combination of leaf rust and Septoria leaf spots also were dramatically reduced with the same treatments in North Dakota. Stratego, a Novartis product, and BAS 500, a BASF product, are not yet registered for wheat, although a request for a special exemption for use of Folicur will again be made in 2000. Additional cooperative fungicide trials across states and wheat classes will be made in 2000.

Application techniques: Progress also has been made on application techniques to improve coverage of wheat heads and increase disease control. Preliminary tests in North Dakota had indicated that the use of flat fan nozzles with medium droplet sizes (XR8002) directed straight downward from the spray boom were not delivering much of the spray to the head, but instead to the leaves or ground (Hofman et al., 1998). Initial spray studies were established with nozzles that directed the spray at an angle toward the grain head, plus provided a smaller droplet size. Spray coverage has been increased dramatically with this angled technique with various nozzles, including an angled air assist type nozzle (Lukach, J., et al., 1999). Disease control has been improved proportionately, as well. Field and greenhouse studies have also indicated that increased water volume generally increases spray coverage and disease control. Studies with aerial application of fungicides has indicated that improved results are achieved with application in early morning or late evening, when natural dews are present to provide some additional water for better distribution of the fungicide across the wheat head. Further studies with aerial application are planned for 2000.

Summary

All of the efforts in studying fungicides for FHB control and in improving methods of delivery are leading to positive results. New products with increased flexibility of

application timing have become available in recent years, either through special registrations or full registrations. Additional products are being tested and look very promising. Studies with application techniques have demonstrated ways to improve head coverage and disease control, without sacrificing leaf disease control, as well. These results give wheat growers more opportunities for combating this disease and provide applicators with opportunities for economically modifying their equipment for improved control.

References Cited

Bai, G., and Shaner, G. 1994. Scab of wheat: prospects for control. Plant Disease 78:760-766.

Hofman, V., McMullen, M., Gregoire, T., and Wiersma, J. 1998. Application of fungicides for suppression of Fusarium head blight (scab). North Dakota State University Extension Circular AE-1148. 2p.

Lukach, J., Halley, S., and Gregoire, T. 1999. Effect of fungicides and sprayer nozzles on control of Fusarium head blight in wheat, 1998. Fungicide and Nematicide Tests 54:332-333.

Mauler-Machnik, A., and Zahn, K. 1994. Ear fusarioses in wheat – new findings on their epidemiology and control with Folicur. Pflanzenschuz-Nachricten Bayer 47:129-155.

McMullen, M., Jones, R., Gallenburg, D. 1997. Scab of wheat and barley – a reemerging disease of devastating impact. Plant Disease 81:

McMullen, M., Milus, G., and Prom, L. 1999. 1999 Fungicide trials to identify products effective against Fusarium head blight in wheat. Proceedings of the US Wheat and Barley Scab Initiative Forum, Sioux Falls, SD: in press.

McMullen, M., Halley, S., Pederson, J., Lukach, J., Endres, and Schatz, B. 1999. Evaluation of fungicides for suppression of head and leaf diseases in wheat: summary of 1999 ND uniform trials. ND 2000 Crop Production Guide. Extension Rprt.: in press.

Mesterhazy, A., and Bartok, T. 1996. Control of Fusarium head blight of wheat by fungicides and its effect on the toxin contamination of the grains. Pflanzenschutz-Nachrichten Bayer 49:187-205.

Milus, E. A., and Parsons, C.E. 1994. Evaluatien of foliar fungicides for controlling Fusarium head blight of wheat. Plant Disease 78:679-699.

Stack, R. W. 1999. Return of an old problem – Fusarium head blight of small grains. APSnet Feature Article, May 1999, www.scisoc.org/feature/FHB/Top-html.

Residue Management and Fusarium Head Blight of Wheat

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Fusarium head blight (FHB) of wheat has become a major problem in the eastern and midwestern United States. Epidemics have caused extensive damage through direct losses in yield and test weight and by price discounting due to the presence of Fusarium-damaged kernels and their associated mycotoxins, mainly the trichothecene deoxynivalenol.

The *Fusarium* species which incite FHB are all capable of surviving saprophytically on host debris. *F. graminearum* occurs on a wide range of gramineous hosts, although FHB epidemics are generally considered to originate from inoculum associated with host residues. There is considerable evidence that continuous wheat cropping (Snyder and Nash, 1968) or wheat following corn (Sutton, 1982) in a rotation system significantly increases the incidence and severity of FHB. Potential inoculum for FHB is reported to be mainly ascospores produced on host residues that have remained at or above the soil surface. Practices for suppressing initial inoculum, especially rotation of wheat and corn with non-host crops and plowing infested residues, have long been recommended for managing FHB (Hoffer et al., 1918).

Despite our knowledge that crop residues at the soil surface serve as a principal source of inoculum, increases in conservation tillage throughout the United States have been significant over the past decade (Anon., 1995). Conservation tillage systems involve leaving all or part of the crop residue on the soil surface after harvest in efforts to reduce soil erosion caused by wind and water runoff. In 1990, federal farm legislation required the adoption of conservation plans that mandated a minimal requirement of 30% residue cover at the time of crop emergence for land classified as being subject to soil erosion (Anon., 1991). This legislation, following extreme drought conditions in the Upper Midwest in 1988, led to the rapid adoption of reduced tillage practices throughout much of the region. These practices contributed to significant increases in crop residues across much of the small grains production area in Minnesota.

The devastating epidemics of FHB in the Upper Midwest raised questions about the causes leading to these outbreaks and generated new interest in developing effective management strategies. Genetic immunity to FHB in wheat has not been reported, and given the incomplete resistance and a lack of highly effective fungicides, effective disease management will likely rely on an integrated management system using a number of control options. Reducing inoculum of *F. graminearum* in host debris, and other reservoirs, may be a key to disease management.

Studies to examine the effect of previous crop residues and tillage practices on FHB of wheat were undertaken. Fusarium head blight was monitored in plots of the FHB susceptible spring wheat cultivar Norm following previous crops of corn, wheat, and soybeans in 1995, 1996 and 1997. Moldboard plow, chisel plow, and no-till tillage treatments, were imposed perpendicular to crop strips to establish a range of residue levels in each of the previous crop residues. Fusarium head blight incidence and severity were greatest when wheat followed corn and least when wheat followed soybeans. Incidence and severity were lower in moldboard plowed plots than either chisel plowed or no-till plots, although differences among chisel plow and no-till treatments were not apparent. Yields of wheat were approximately 15% lower in plots where wheat followed corn or wheat than in wheat following soybeans and were 10% greater in moldboard plowed plots than in either chisel plowed or no-till treatments. The deoxynivalenol (DON) content of harvested grain was significantly correlated with FHB incidence and severity. The DON level in wheat following soybeans, averaged across tillage treatments, was 25% lower than in wheat following wheat, and 50% of the level in wheat following corn. These findings suggest that changes in regional tillage practices, principally the move toward conservation tillage and reduced-till systems, contributed to the recent FHB epidemics in the Upper Midwest. As differences in the type and quantity of crop residues in small plots impacted disease development, it is likely that local sources of inoculum, such as those within a grower's field, contribute directly to the inoculum load and disease potential. The implications of these findings are that selection of cultural practices aimed to reduce inoculum borne residues will assist in the control of FHB.

Additional studies have been undertaken to examine the survival of *F. graminearum* in relation to wheat residue decomposition. Crop residues of the FHB susceptible spring wheat cultivar Russ, consisting primarily of nodes and internodes, were placed on the soil surface and at 10 and 20 cm depths in soil which had been chisel plowed. Residue was also placed 20 cm below the surface in soil which had been moldboard plowed. The trial was established in October 1997 and residue was recovered monthly between April and November in 1998 and 1999. Dry matter determinations on residue samples placed below the soil surface indicated that 60 % of dry matter was lost in the first year and up to 80 % of dry matter was lost after two years. There appeared little difference in the rate of residue decomposition between residue in moldboard plowed treatments or in chisel plowed treatments at either the 10 or 20 cm depths. Decomposition of residue placed at the soil surface was slower than that of the buried residue with approximately 30% and 60% of dry matter lost after one and two years, respectively. Survival of *F. graminearum* in residue was determined on node tissue. Recovered nodes were separated from other residues, surface disinfected, and plated onto pentachloronitrobenzene agar for the isolation of *Fusarium*. *F. graminearum* was identified following transfer to carnation leaf piece agar. *F. graminearum* was recovered from residue in all treatments at each sampling time for the duration of the study. Recovery of *F. graminearum* diminished over the first year with the percent of infected nodes dropping from ca. 90 % to between 50 and 80 %. Survival of *F. graminearum* in surface residues was higher than in buried residue and appears to be related to the rate of residue decomposition. While data on the survival of *F. graminearum* from the second year of the trial is not yet analyzed it is

evident that *F. graminearum* is capable of surviving in wheat residue on the soil surface for over two years. The ability of these residues to support the production of mature perithecia and ascospores was also confirmed.

Literature Cited

Anonymous. 1991. Conservation in the 1990 Farm Bill. United States Department of Agriculture, FACTA 2, 3 p.

Anonymous. 1995. Conservation Impact, 13 (9). Conserv. Technol. Information Ctr. Newsl., West Lafayette, IN.

Hoffer, G. N., Johnson, A. G., and Atanasoff, D. 1918. Corn-rootrot and wheatscab. Journal of Agricultural Research 13:611-612.

Snyder, W. C., and Nash, S. M. 1968. Relative incidence of Fusarium pathogens of cereals in rotation plots at Rothamsted. Trans. Br. Mycol. Soc. 51:417-425.

Sutton, J. C. 1982. Epidemiology of wheat head blight and maize ear rot caused by Fusarium graminearum. Can. J. Plant Pathol. 4:195-209.

Effects and Survival of Seed-borne *Fusarium*

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Introduction: Fusarium head blight (FHB) has become the most damaging disease of wheat in the prairies since the major epidemic of 1993. After several years of environmental conditions that favour development of FHB, and observing its occurrence in more westerly areas of Manitoba and south-eastern Saskatchewan, we are in urgent need of understanding the means by which the disease may be spread, including dispersal via infected seed. Can we inadvertently spread fusarium head blight (FHB) by sending infected seed to FHB-free areas of the prairies? Does a crop grown from FHB-infested seed suffer higher levels of FHB?

Current recommendations to prairie producers faced with a wheat crop infected with fusarium head blight (FHB) include removing as much of the light weight damaged grain as possible by opening the louvres and blowing it out the back of the combine. The ultimate fate of seed-borne *Fusarium* (mostly *F. graminearum* , the predominant species causing FHB in North America) is a concern. It is not known how long fusarium damaged kernels (FDK) remain intact under prairie conditions before they decompose, and more importantly how long the fungus remains viable on the seed lying either on the soil surface, or buried as a result of cultivation. If the fungus remains viable on FDK for any length of time what effect does that have on succeeding crops?

Three related studies were initiated to try to answer these questions.

1. The role of infected seed in the development of FHB. Study sites included Glenlea, MB, Brandon, MB, Swift Current, SK, and Lethbridge, AB. Seed samples of spring wheat cultivars AC Domain and Glenlea were rated for percent *Fusarium* infection and mixed in the following ratios with healthy seed of the same variety: 100:0, 90:10, 75:25, 50:50, 25:75, 0:100, healthy:infected, respectively. Plots were four rows by 3 m. The experiment was planted in a randomized complete block design with 6 replications. Emergence was measured by counting plants at the 2-3 leaf stage in 0.5 m of each of 4 rows (2 m in total). A measure of stand establishment was made by counting the number of tillers in the same 0.5 m of each row (2 m in total) after heading. To examine leaf tissue for presence of *F. graminearum*, 10 leaves per plot were sampled from the canopy at Time 1 (GS 15-30, before end of tillering), upper and lower canopy at Time 2 (GS 37 flag leaf still rolled) and lower, mid and upper canopies at Time 3 (GS 65 wheat in flower). Plots were scored 21 days after anthesis to give a visual rating for FHB.

Emergence and stand establishment (tillering) data are presented in Table 1. As proportions of infected seed increased, emergence and tillering decreased, but the reduction in tillering was not as high as the reduction in emergence. When the varieties were examined separately, it was found that emergence of cv. Glenlea was higher than cv. AC Domain, but AC Domain compensated for poor emergence with more tillers. Examination of leaf tissue is ongoing. In 1998, the leaves sampled at Time 1 from Brandon and Swift Current showed only very low levels of *Fusarium graminearum*, and

no *F. graminearum* was found at Glenlea, MB. Isolations of *F. graminearum* and FHB severity were not different among treatments (Table 2).

Table 1. Least squares means for emergence and tillering of spring wheat infected with fusarium head blight.

Treatment- ratio of healthy:infected seed	Emergence per 2 m row	Tillering per 2 m row
100:0	63.8 a [1]	65.7 a[2]
90:10	63.2 a	62.9 ab
75:25	56.8 b	66.3 a
50:50	49.8 c	59.3 bc
25:75	44.0 d	55.6 c
0:100	38.2 e	56.0 c
Lsd	4.3	4.1

[1] Emergence at 2-3 leaf growth stage

[2] Numbers of tillers after heading

Numbers within columns followed by the same letter are not significantly different at $P \leq 0.05$.

Table 2. Fusarium head blight severity across treatments at Glenlea, Manitoba - 1998-1999.

Treatment- ratio of healthy:infected seed	FHB severity			
	AC Domain		Glenlea	
	1998	1999	1998	1999
100:0	20.3	7.0	20.8	13.0
90:10	11.8	7.8	11.3	8.8
75:25	10.5	7.0	9.5	9.3
50:50	16.8	10.0	13.0	14.5
25:75	13.8	8.3	13.0	14.3
0:100	13.0	12.8	10.5	10.8

2. Duration of survival of *F. graminearum* on FDK. FDK were separated from FHB-contaminated seed samples of the two spring wheat cultivars, Roblin and AC Domain and placed into small nylon mesh bags. The bags were left exposed on the soil surface or buried at 5 or 10 cm. The study was set up in a randomized complete block design with 4 replicates. In the field, each bag containing 50 FDK represented a replicate. Sampling

took place at 6 and 12 months and will continue for a second year (at 6 monthly intervals) to determine the duration of survival of *F. graminearum*. Under controlled conditions bags of 25 FDK were left on the surface or buried at 5 cm in sterile or non-sterile soil and exposed to a constant temperature of 20EC, 5EC, or -10EC. At 6 and 12 months there was no significant difference in numbers of FDK from which *F. graminearum* was isolated. At the first sampling period, after one fall and winter, seeds were not different in mass from when the experiment was established. After 12 months, the seeds that were buried 10 cm in the field were partially decomposed and some seeds were lost upon retrieval. However, *F. graminearum* was isolated from all retrieved seeds. Survival ranged from 87-100 % over all treatments in both the field and under controlled conditions after 12 months. Greatest loss of viability (13%) occurred under controlled condition at 20EC in sterile and non-sterile soil.

3. Susceptibility of crops to soil-borne *F. graminearum*. The third study was established to determine whether *F. graminearum* isolated from wheat heads can transmit disease to roots or underground parts of crops used in rotations. The crop spp. tested included wheat, barley, oat, rye, triticale, canaryseed, *Brassica napus*, *B. rapa*, *B. juncea*, bean, pea, lentil and chickpea. One floret or FDK of Roblin wheat infected with a single *F. graminearum* isolate, was placed adjacent to seeds of each crop at seeding. Emergence was counted, and disease symptoms on sub-crown internodes or hypocotyls were scored 4 weeks later. Emergence was significantly reduced in all crop spp. except peas and *Brassica* spp.(Fig. 1). Infections of subcrown internodes or hypocotyls were high in wheat, barley, pea, chickpea and lentil, but moderate in rye, oat, triticale and canaryseed (Fig 2A, B). Emergence and root infections were negatively correlated in all crop species except for peas. Emergence of peas was not significantly reduced when planted beside *F. graminearum*-infected wheat kernels or florets, but suffered high levels of disease on the hypocotyls. *Fusarium graminearum* was shown to infect several crop species and these results may have significant implications on crop rotation as a strategy for disease management.

Conclusions:

It appears unlikely that planting seed infected with *F. graminearum* directly causes increased levels of FHB in wheat. Disease severity was no different in plots planted with 100% fusarium-infected grain sample and plots planted with 100% fusarium-free seed. However, *F. graminearum* survives on FDK on and in the soil for at least 12 months and may cause reduced emergence and root rot in rotational crops. These aspects of FHB management need to be researched more thoroughly.

Acknowledgments:

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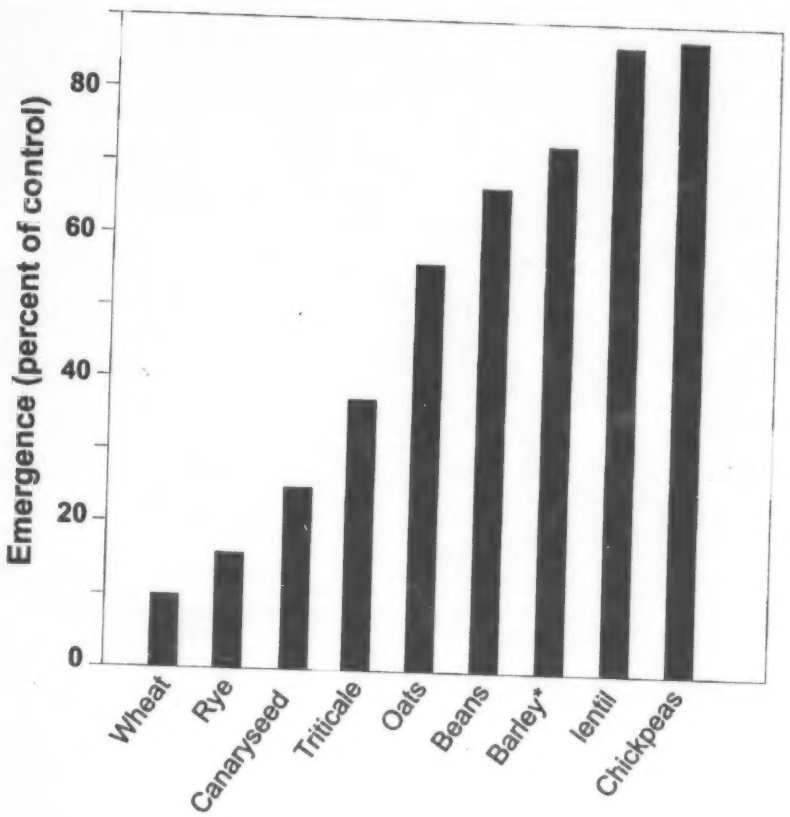

Fig. 1. Emergence of crop species when planted adjacent to fusarium damaged kernels or infected florets

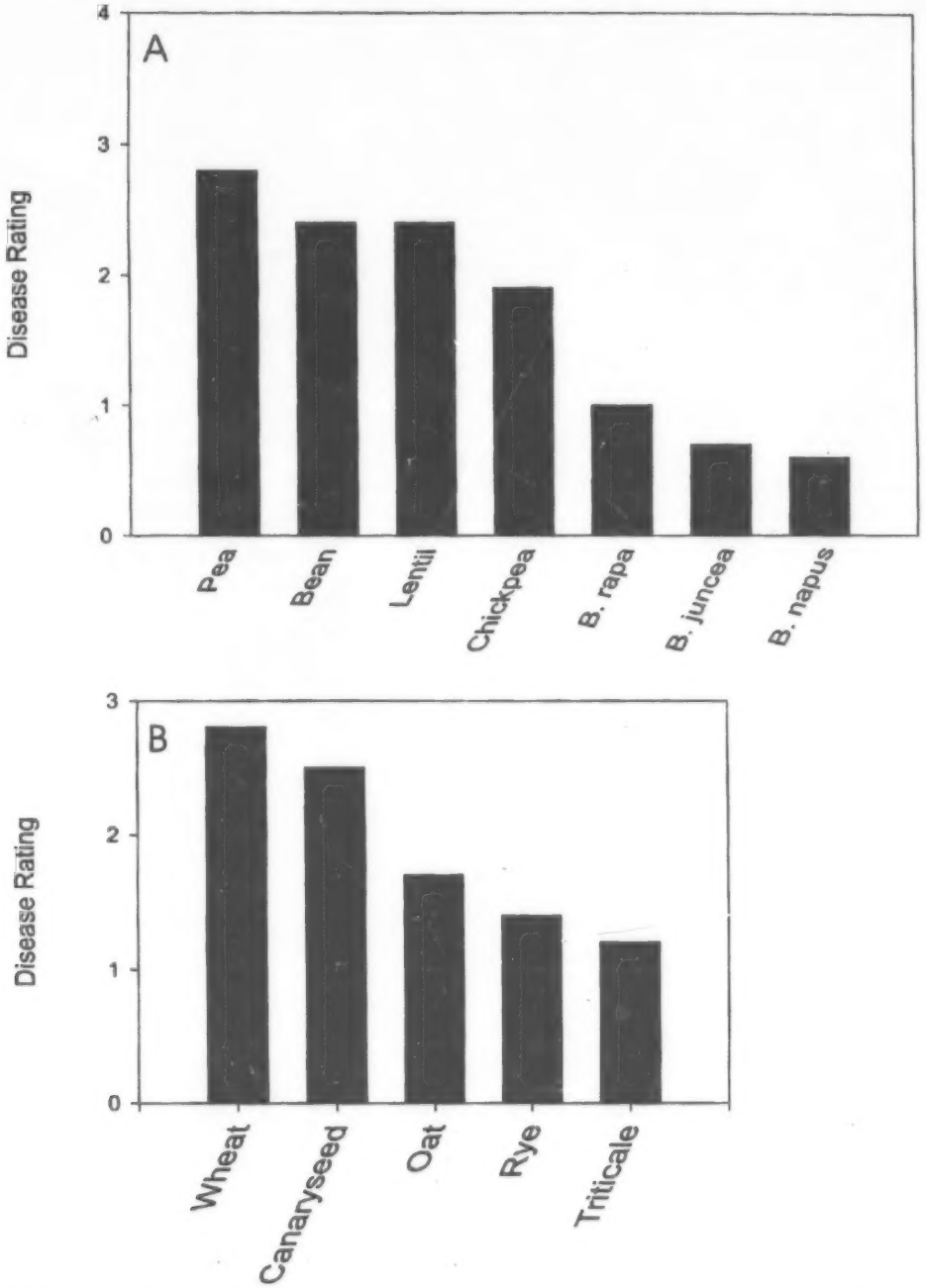

Fig. 2. Root disease of crop species when planted adjacent to fusarium damaged kernels or infected florets. A. Hypocotyls (Hwang et al. 1994. CJPP 16:295-303) B. Subcrown internodes (Ledingham et al. 1973. CPDS 53:112-113).

Population Structure and Genetics of *Gibberella zeae*

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Population Structure

Gibberella zeae is one of the most important plant pathogens in the world (McMullen et al., 1997). As we develop and evaluate control strategies for this intractable pathogen, we must consider the potential for the fungus to evolve countermeasures. Meidaner (1997) thought the risk of adaptation to host resistance by *G. zeae* was low because resistance appears to be quantitatively inherited and no consistent isolate-by-host interaction has been reported. However, some disease resistance quantitative trait loci (QTL) are isolate-specific in other pathosystems (Young, 1996). Since most of our current resistance to scab can be traced back to just a few sources, each containing only a few genes (Van Ginkel et al., 1996), the possibility of adaptation to resistance genes cannot be ignored. Adaptation to chemical or biological control is also possible. Understanding the genetic structure of *G. zeae* populations can help elucidate the roles of population subdivision, gene flow (migration), sexual recombination, mutation, genetic drift, and selection on the evolutionary potential of this pathogen.

Bowden and Leslie (1992) described high levels of genotypic diversity in *G. zeae* in a collection of 24 isolates from Kansas. Each isolate belonged to a different vegetative compatibility group (VCG), which showed that they were all different genotypes. Ouellet and Seifert (1993) reported a relatively low amount of genetic diversity in *G. zeae* in Ontario, but they used a small set of RAPD markers. Bowden and Leslie (1994, 1997), using VCGs, reported high genotypic diversity of *G. zeae* from single wheat heads in Kansas. Schilling et al. (1997) using a large set of RAPD markers, found high genetic diversity within fields and within heads of wheat in Germany. In Korea, Moon et al. (1999) found that each of 53 isolates belonged to different VCGs.

Although VCGs have provided some basic information about populations of *G. zeae*, the technique is ill suited for extensive studies of genotypically diverse populations. VCG data are generated by pair-wise comparisons among all strains. The number of tests increases as the square of the sample size so the number of tests balloons quickly. VCG studies also do not provide allele frequencies (unless the genetic basis of every VCG is known), which are needed to compare populations. Molecular markers are better suited for such studies. RAPD fingerprinting is relatively cheap and easy, but the number of polymorphic bands per reaction may be limited and reproducibility is sometimes questionable. AFLP fingerprinting (Vos et al., 1995) is technically more difficult, but generally produces a large number of polymorphic bands per reaction and has excellent reproducibility.

Zeller, Bowden, and Leslie (unpublished) used AFLPs to determine the genetic diversity of 63 isolates of *G. zeae* from a small (0.5 m x 0.5 m) quadrat in Kansas and 63 isolates

from a similar quadrat in N. Dakota. Using two sets of AFLP primers, 43 polymorphic loci could be scored. Although some haplotypes were detected more than once in each quadrat, the haplotypes in North Dakota were all different from those in Kansas. Identical AFLP haplotypes may represent localized secondary infections. Duplicate haplotypes were censored for further analysis. We determined allele frequencies (presence or absence of band) for each locus in each sample. Nei's (1973) G_{ST} index failed to detect any differences in allele frequencies between the Kansas quadrat and the North Dakota quadrat. Although a few alleles occurred only in one population, all were rare and could not be used to distinguish the populations. Although the sample size was small and the sampled area very restricted, the preliminary conclusion is that populations in Kansas and North Dakota are very similar. This study needs to be expanded to more regions, larger sample sizes, and more markers.

Genetics

Bowden and Leslie (1999) described methods for crossing *G. zeae*. Since *G. zeae* is homothallic, markers are necessary to distinguish selfed from outcrossed perithecia. They found that nitrate nonutilizing (*nit*) mutations were suitable markers and used them to show that strains of *G. zeae* from North America, Asia, and Africa were all interfertile.

Jurgenson, Bowden, and Leslie (unpublished) created a genetic linkage map of *G. zeae* by crossing a strain from Kansas with a strain from Japan. Characteristics of the two parents are presented in Table 1. Ninety-nine *nit+* progeny were selected and analyzed for polymorphisms using AFLP markers. We used thirty-three two-base selective primer pairs and found 1084 polymorphic loci of which 1029 unambiguously segregated into nine linkage groups. We estimated the total map length of the genome is in excess of 2700 cM with an average interval of 2.6 map units between loci. Three linkage groups exhibited a high degree of segregation distortion. Selection of *nit+* progeny accounts for some but not all of the segregation distortion observed.

Table 1. Characteristics of parents of mapping cross.

Characteristic	Z-3639	R-5470
Geographic origin	Kansas	Japan
Original host	Wheat	Barley
Toxin types*	Deoxynivalenol	Nivalenol, Fusarenon-X
Toxin quantity*	High	Low
Aggressiveness	High	Unknown
Perithecial production	Abundant	None
Colony color	Pink	Tan
nit marker	*nit3*	*nit1*

*Information provided by Dr. Ron Plattner, USDA/ARS, Peoria, Illinois.

Development of a genetic map has several benefits. First, knowledge of linkage relationships would improve population genetic studies by removing the bias created by linkage between molecular markers used to monitor genetic recombination, gene flow, mutation, and genetic drift. Second, a genetic map can be used for map-based cloning of

important genes related to virulence, mycotoxin production, competitive ability, sensitivity to fungicides, etc. Third, a genetic map can be used for detecting QTLs controlling traits such as aggressiveness, growth rate, etc. Fourth, a genetic map of *G. zeae* could be compared to other genetic maps of related Ascomycete fungi, such as *G. fujikuroi* (Xu and Leslie, 1996), to study the degree of gene synteny and the evolution of chromosomal organization.

References

1. Bowden, R. L., and Leslie, J. F. 1992. Nitrate-nonutilizing mutants of *Gibberella zeae* (*Fusarium graminearum*) and their use in determining vegetative compatibility. Exp. Mycol. 16:308-315.
2. Bowden, R. L., and Leslie, J. F. 1994. Diversity of *Gibberella zeae* at small spatial scales. Phytopathology 84:1140 (abstract)
3. Bowden, R.L., and Leslie, J.F. 1997. Diversity and sexuality in *Gibberella zeae*. Pages 35-39 in: Fusarium Head Scab: Global Status and Future Prospects. H.J.Dubin, L. Gilchrist, J. Reeves, and A. McNab, eds. Mexico, D.F.:CIMMYT.
4. Bowden, R. L. and Leslie, J. F. 1999. Sexual recombination in *Gibberella zeae*. Phytopathology 89:182-188.
5. McMullen, M. P., Jones, R., and Gallenberg, D. 1997. Scab of wheat and barley: a re-emerging disease of devastating impact. Plant Dis. 81:1340-1348.
6. Meidaner, T. 1997. Breeding wheat and rye for resistance to Fusarium diseases. Plant Breeding 116:201-220.
7. Moon, J-H., Lee, Y-H., Lee, Y-W, Moon, J.H,. Lee, Y.H., Lee, Y.W. 1999. Vegetative compatibility groups in *Fusarium graminearum* isolates from corn and barley in Korea. Plant-Pathology-Journal.15: 53-56.
8. Nei, M. 1973. Analysis of gene diversity in subdivided populations. Proc. Natl. Acad. Sci. 70:3321-3323.
9. Ouellet, T. and Seifert,K. A. 1993. Genetic characterization of *Fusarium graminearum* strains using APD and PCR amplification. Phytopathology 83:1003-1007.
10. Schilling, A. G., Miedaner, T., and Geiger, H. H. 1997. Molecular variation and genetic structure in field populations of *Fusarium* species causing head blight in wheat. Cer. Res. Comm. 25:549-554.
11. Van Ginkel, M. Van Der Schaar, W., Yang, Z. P., and Rajaram, S. 1996. Inheritance of resistance to scab in two wheat cultivars from Brazil and China. Plant Dis. 80:863-867.
12. Vos, P., Hogers, R., Bleeker, M., Reijans, M., van de Lee, T., Hornes, M., Frijters, A., Pot, J., Peleman, J., Kuiper, M., and Zabeau, M. 1995. AFLP: a new technique for DNA fingerprinting. Nucleic Acids Res. 23:4407-4414.
13. Xu, J.-R., and Leslie, J. F. 1996. A genetic map of *Gibberella fujikuroi* mating population A (*Fusarium moniliforme*). Genetics 143:175-189.
14. Young, N. D. 1996. QTL mapping and quantitative disease resistance in plants. Ann. Rev. Phytopathol. 34:479-501.

Recent Progress in Molecular Strategies to Improve the Fusarium Tolerance of Plants

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The mycotoxin deoxynivalenol (DON) produced by *Fusarium graminearum* is a potent inhibitor of eukaryotic protein synthesis and is believed to play a role in fungal pathogenesis on cereal crops world-wide. The putative site of action of this molecule has been postulated to be the 60S ribosomal protein L3 (RPL3), an integral component of the protein synthesis machinery of all eukaryotic cells. We have modified a rice (*Oryza sativa* L.) cDNA encoding the ribosomal protein L3 so that amino acid residue 258 is changed from tryptophan to cysteine, a change which is believed to confer resistance to similar mycotoxins in yeast. Both the modified and unmodified versions of the rice *Rpl3* genes were introduced into two species of tobacco (*Nicotiana tabacum*, *N. debneyi*) by *Agrobacterium tumefasciens* co-cultivation and expressed under the control of the cauliflower mosaic virus 35S promoter. When cells, tissues, and protoplasts of these transgenic tobacco plants were compared for growth in the presence of DON, a significant difference in growth rate and the ability to undergo differentiation was observed among those plants expressing the modified version of *Rpl3* (*Rpl3:c258*), compared to those expressing the wild-type rice *Rpl3* gene. These results indicate a possible mechanism of host plant resistance to the fungal pathogen F. graminearum among the susceptible cereal species (corn, wheat, barley, rice, oats) based on the expression of modified *Rpl3* genes. This strategy is currently being evaluated in corn and wheat. Other strategies to improve host plant resistance to this fungal pathogen include the cloning and expression of single chain fragment variable (scfv) regions of an anti-DON monoclonal antibody, and the search for novel, induced, resistance genes early in the infection process of maize plants. Funding support of the Ontario Corn Producers' Association , Ontario Research Enhancement Program, Canadian Adaptation Council, and the AAFC Matching Investment Initiative is gratefully acknowledged.

Sources of Resistance to FHB

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Sumai3 has been recognized as one of the best sources of tolerance to FHB in terms of lower floret infection and lower DON deposition in the seed. FHB Breeding programs in many programs internationally how successfully transferred this "resistance" into advanced breeding material. However, the Sumai3 level of resistance will still suffer 20% floret infection in epiphytotic nurseries. Therefore there is a need to identify additional sources of resistance with which to enhance the Sumais level of resistance and to broaden the gene pool of FHB tolerant alleles.

In this overview the variability for FHB tolerance was examined in a. mapping populations, b. wheat accessions, c. secondary and d. tertiary gene pool of wheat.

Our main population consists of doubled haploid (DH) lines derived from a hybrid between Sumai3 and HY368. The best segregates in this population showed only a nominal improvement over sumai3 and several lines were identified with relatively high levels of floret infection but low DON levels. The reciprocal combinations were not detected.

Our alternate mapping population consists of 101 DH lines derived from a cross between the cultivars Wuhan and Maringa from China and Brazil respectively and each showing a reasonable level of FHB resistance. A number of lines exceed the parents in levels of FHB resistance and one line combines reasonable FHB resistance with a fairly high level of BYDV resistance inherited from Maringa (phenotyping done by Steve Haber). We intend to develop markers for both of these segregating traits.

b. Wheat accessions

We have acquired FHB resistance in improved plant types from several sources e.g. Europe, CIMMYT, Brazil, China. Their allelic relationships with Sumai3 is not known as yet. These have been intercrossed in 40 3 and 4 way combinations and F_2 progeny grown and inoculated in field plots. Although highly susceptible segregates were observed in these populations it is not known if this means the segregation of different FHB resistance genes. Selections from these nurseries are currently growing as F_3 hills in a winter nursery. They will be re-examined as F_4 rows in a FHB nursery next year. Accessions from Japan also provide high levels of FHB resistance especially such cultivars as Shinchunaga.

Very intensive screening efforts are underway in U.S. labs of landraces of wheat acquired from areas of the world where FHB is endemic, e.g. China, Japan, Italy, Brazil. For example Ann McKendry (Missouri) has recently screened 937 landraces of winter wheat and found 38 that appeared to be resistant and another 150 that will be retested. She intends to screen another 1000 accessions that originated in the Balkan countries. Greg Shaner and Herb Ohm (Purdue) have been screening winter wheat accessions particularly from the Far East on a continuing basis and have identified additional promising accessions.

Miller, Stack and Joppa (Proc. 9th IWGS3: 293- , 1998) have screened 400 *T.*

dicoccoides accessions and found heritable variation, the best of which has been crossed into the best durum lines. J. Gilbert and J. Clarke have also identified promising *T. dicoccoides* accessions from their screening efforts.

c. Secondary gene pool

The secondary gene pool of wheat represents the *Aegilops* species. For most of these species embryo rescue will be required to obtain hybrids with wheat; the hybrids will be sterile but straight forward backcrossing will be successful in restoring fertility and introgressing the traits in question. Several hundred accessions of each of *T. monococcum*, *Ae. speltoides* and *Ae. squarrosa* have now been screened by ourselves, Yu Jin (South Dakota) and Paul Murphy (North Carolina). Very few promising accessions have been identified to date. This may be due to the fact that not many of these species grow in areas where FHB is endemic and thus have not evolved resistance mechanisms. In our screening we have progeny from several *T. timopheevi* (AAGG) lines that had resistance to *Septoria* tan spot and some tolerance to FHB. BC_2F_1 progeny will be available for spring planting and evaluation in 2000.

d. Tertiary gene pools of wheat

The tertiary gene pool of wheat includes such species as *Secale, Thinopyrum, Elymus, Leymus, Agropyron, Dasypyrum*, and *Hordeum*. Resistance has been detected in all of the above but introgression into wheat will be quite slow and laborious. Crossability with wheat will be low, necessitating the use of embryo rescue. Chromosome pairing between parental genomes will likely be negligible so that Ph mutants of wheat will need to be used to make the first cross (and even the first backcross). The other alternative is to backcross onto the intergeneric hybrids to produce the respective addition lines, then induce recombination by Ph mutants, irradiation or callus cultures. For introgressions by these methods it is important to involve homologous chromosomes and introgress the smallest possible segments to minimize linkage drag.

There are a number of species collected in FHB endemic areas of China and Japan that have virtual immunity to FHB. One such species is *Elymus humidus* (SHY genome) that showed no symptoms from either point and spray inoculations under our conditions where the susceptible check (Roblin) was totally infected after 10 days. The resistance from another two such species, *Roegneria komoji* and *R. ciliaris* has already been introgressed into wheat by scientists in China. Other species with such levels of resistance include *E. giganteus, E. filvosa, L. racemosus, R. stenostachys*, and *R. stricta*.

In our own screening, we have identified resistance in addition lines derived from *S. cereale* (4, 5, 7), *H. chilense* (1 and 2), *H. californicum* (4), *Th. intermedium* (3, 7), *D. villosum* (6) and *Agropyron cristatum*. In all cases, seed of the addition lines has been irradiated and progenies as being screened in field FHB nurseries. Other Triticeae species identified as having FHB resistance include *Th. distichum* and *L. multicaulis* (Comeau), *Th. junceiforme* and *L. elongatum* (Jauhar).

The above overview indicates that numerous accessions with FHB resistance have now been detected in primary, secondary and tertiary gene pools of wheat. The next important task will be devising methods and testing these materials to determine their

allelic relationships regarding FHB resistance.

Multiplex PCR Kits for Pyramiding FHB Resistance Genes in Wheat

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For the complex genetic system of FHB resistance in wheat, three additive resistance genes on separate chromosomes have been identified using a doubled haploid segregating population. Each resistance gene contributes about 25-40% of the total resistance found in Sumai3. User-friendly and co-dominant markers linked to all three resistance loci have been developed. All three genes can be detected simultaneously by multiplexing. Seed material can be tested thus eliminating the lengthy growing period prior to conventional inoculation testing. The type II (spreading) and type I (initial infection) resistance genes have been tentatively identified .

Multiple markers for each resistance gene allowed the graphical genotyping of multiple alleles flanking these genes. Graphical genotyping of numerous resistant and susceptible lines will determine if these markers are informative in these lines. The co-segregation of the FHB resistant phenotype and these markers has been shown for at least 5 generations thus the markers can track the resistance genes. The markers are being used by breeders to pyramid FHB resistance genes into elite cultivars.

Breakout Group Reports

1) PRIORITIES AND NEEDS

Chair: James H. Helm

The purpose of the breakout groups was to look at the Fusarium Head Blight problem from four distinct focus groups , Breeding and Genetics, Prevention of Spread, Industry Issues and Biotechnology, to determine the most significant priorities that will help us meet the challenge. The reports from each of the groups are presented by the facilitators of the groups.

BREEDING AND GENETICS

Facilitator: Les Shugar
Assistant Facilitator and Secretary - A. Comeau

Quote of the day:

"...Whenever a worrisome outcome seems likely but data seems too sparse for firm conclusions, scientists need to work hard to fill the void. They need to plant the right experiments, gather the needed data and publicize the results in both public and specialist media. And the public needs to provide the funds - the tax dollars - to support this work, since most of it will need to be done by scientists in public institutions." [2]

Discussions were organized around a general plan that was adopted by the group. We first talked of the difficulties of phenotyping FHB reaction, a step that is more difficult for barley but also real in wheat. Priority to be given to species was not controversial; the key species is *F. graminearum*, and other species are understood to deserve some attention where justified.
The importance of applying attention to crop residues in breeding work was discussed and questions were raised about the effect of residues on prevalence of species or isolates and about the effect of residues on gene exchange (sexuality) between Fusarium VCG (vegetative compatibility groups). The need to understand the plasticity of pathogen behavior was mentioned together with a need for surveys that include virulence data. Breeding methodologies tended to include both conventional and haploid breeding approaches with some male sterile work in barley (Therrien). Resistance type 1 was desired by 6 breeders; type 2 by 3 breeders; a mix of types by 3 breeders.
Morphological criteria that may correlate to resistance were mentioned as worthy of attention. In barley, flowering in the boot, absence of lateral florets in 2-row, are

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[2]Duvick DN 1999, Consequences of classical plant breeding for pest resistance. In P.L. Traynor, JH Westwood, eds.Virginia Polytech. Blacksburg VA. Ecological effects of pest resistance in managed ecosystems. Proc. Workshop

examples of criteria. As to whether DON is better than symptoms; it was noted that genes might differ.

It was mentioned that phenotyping from symptoms requires constant presence in the field during the evolution of symptoms, as these may change very rapidly.
Are genes "major" or "minor"? Major genes are poorly known at this point, some question that they exist; others feel they have seen 1:2:1 ratios in recent studies where 2 dominant genes are suspected in wheat crosses (Shugar). There is little Canadian data on this topic to date.

Resistance sustainability was seen as a future question rather than a current one. As to pleiotropic effects, these were viewed as needing to be dealt with when they are confirmed.

The Winnipeg program relies on 3 markers that seem to fit both type 1 and 2 classifications; these markers account for 50% of variability. There is one main group doing the marker assisted work in wheat (Winnipeg), with smaller initiatives elsewhere. This group relies on symptoms, DON, infected florets as criteria to correlate with markers. Markers are ready to use in wheat only; in barley and corn they are not.

The matter of sharing gene marker information was raised; the willingness to share germplasm and markers could be an issue as this is not so automatic now as it was 25 years ago but public breeders expressed willingness to share, yet details of how this gets done cannot be defined without further discussion.

Briggs mentions that little progress can be expected with approx. 5000 lines evaluated per year per program for multiple traits. Winnipeg may handle 400 lines per cross to handle larger number of traits. Convergent methods can also reduce number of dh lines needed per cross. Use of isogenic lines is a possibility but not really explored yet. Geoff Hughes mentioned the importance of testing methods.

The possible impact of information from other disciplines was mentioned. For example, should breeders assess their cultivars under extra pressure from poor management that increases disease or under integrated management that decreases it?

Priorities were spelled out and priorized as follows:

PRIORITIES

1. Genetics of Fusarium resistance is more complex than expected; this raises the population number that must be assessed for yield progress. Higher screening capacity is the no. 1 priority.
2. When available, probes (genetic markers) may reduce costs of breeding significantly; probes, and ways to implement their use are a major priority.

3. Extra research investments are needed and the scientists have no mandate and little time to promote research needs. It was approved that as mechanisms to deal with an exceptional situation are non-existent, the approach of the medical research groups, for example hiring lobbyists, must be supported as a major priority. Society stakeholders (socio-economic, environmental [others] groups) should understand situation-needs and join together to help drive the initiative directly to provincial and federal political representatives.
4a. Considering the difficulties of phenotyping, there is a priority for in-depth research in Fusarium to understand host-parasite interactions and to understand in all the fine details all the events that relate to the infection, from spore germination to tissue invasion, symptom and toxin evolution.
4b (ex aequo). To further pinpoint breeding targets, further understanding of epidemiology and integrated management is a priority.

PREVENTION OF SPREAD

Facilitator: Myriam Fernandez

A brief overview of the Fusarium head blight (FHB) situation in Saskatchewan and Alberta was given by Myriam Fernandez and Kelly Turkington, respectively. FHB and *F. graminearum* continue being localized mostly in the eastern and northern part of Saskatchewan. There has been an increase in the number of fields with FHB in non-irrigated areas of Alberta over the last few years, although the incidence of this disease in the province remains low. *F. graminearum* has been declared a pest under the Alberta Pest Act. The potential implications of this might include recommendations regarding the use of pathogen-free seed.

Of mutual concern to western Saskatchewan and Alberta is the possible continued spread of FHB if environmental conditions remain favourable for the development of this disease. The following issues were identified as playing a role in the prevention of the further spread of FHB in western Canada:

Movement of seed - Controlling the movement of seed from areas affected by FHB was seen as an important strategy in helping to delay the spread of *F. graminearum* into uninfected areas until more resistant varieties are available. Using seed from areas where FHB is well established, even if the seed is symptomless, increases the risk of introducing *F. graminearum* into unaffected areas. It is important to make producers aware of the importance of using clean seed to prevent the further spread of FHB. To this end we should recommend that the seed be tested for the presence of *F. graminearum*, rather than relying on visual symptoms. The efficacy of seed treatments in preventing the spread of the pathogen was questioned, and it was felt that more data was needed in this regard. The possible introduction of the fungus via infected feed grain is also of concern in Alberta. There was also some discussion that controlling the movement of seed may not be overly effective in keeping the fungus out of uninfected areas. Infected seed might not be the primary way of spreading this disease.

Root and crown infections - There was some discussion regarding the possible implications of root and crown infections by *F. graminearum* in the subsequent development of FHB. Survival of the pathogen in underground plant tissues might play an important role in the epidemiology of FHB.

Agronomic practices - the effect of tillage and crop rotation in the development of FHB has not been conclusively determined. It has been difficult to determine the impact of these practices, particularly in years favourable to the development of FHB. Factors such as survival of the pathogen in nonhost residues and inoculum from neighbouring fields or grassy weeds might confound any rotational effects. More research on the effect of agronomic practices on FHB is needed in western Canada. These should be seen not only in terms of strategies to restrict the development of FHB in areas at risk but also to reduce the impact of this disease in areas where the pathogen is well established.

Cultivar - the use of cultivars more tolerant to FHB should be emphasized.

Chemical control - We need to prove that fungicide management is effective in controlling FHB and economical. A forecasting system should be developed to help producers decide whether to spray.

Biocontrol - The need to do research on biocontrol of residue-borne *F. graminearum* was also identified.

It was concluded that we cannot look at all the issues possibly affecting the spread of FHB in western Canada in isolation but need to develop an integrated approach. Developing recommendations to limit the spread of FHB is important not only until we have cultivars with better genetic resistance, but strategies to keep inoculum levels low should be regarded as a long term approach to improve crop protection.

INDUSTRY ISSUES

Facilitator: Karen Dupchak

Malting

Most malting companies, including the largest, will not accept barley with detectable levels of DON. The method used by these companies has a detection limit of 0.5ppm. This requirement is based on image/perception rather than scientific rationale. There was considerable discussion as to what caused the gushing. It is recognized that DON itself does not, but that other compounds, possibly polysaccharides produced by the fungus, may be involved. Antibodies to *F. graminearum* polysaccharides are being used by a Danish brewery for assessing the risk of gushing. Work on this issue is being done in Europe and at the North Dakota State University. These should be consulted before further studies are attempted here. It was then suggested that determining the amount of fungal mass that resulted in gushing was important. This was ruled out by members of

the malting industry who indicated that no level would be acceptable due to the importance of maintaining a pure, uncontaminated image for their product.

NEEDS/PRIORITIES None

DON Testing

The reliability of DON testing and the lack of a check sample program was discussed. The American Association of Cereal Chemists has just initiated such a program for American laboratories. It was agreed that such a service should be provided to Canadian laboratories. Tom Nowicki indicated the Canadian Grain Commission may be able to provide such a service.

There was also discussion about DON testing methodology. David Miller mentioned that FAO has shifted away from research into immunoassays for mycotoxin testing, feeling that development of this methodology has gone as far as it can. They are now recommending the use of high performance TLC (thin layer chromatography). Image analysis and NIR are other methods being studied for DON analysis and measurement of fusarium damaged kernels in grain samples.

NEEDS/PRIORITIES To institute a check sample program for Canadian laboratories testing for DON immediately.

Detoxification

With the exception of the feed industry, the addition of a detoxifying agent, if in fact one exists, is not an option for dealing with DON grain. No federal regulations, anywhere in the world, exist to allow the use of such a product in food grade products. The consensus of the group was that this would never be an option for food grade products.

NEEDS/PRIORITIES The University of Guelph is doing some work with detoxification agents which may be beneficial for the feed industry. This work should continue.

Prevention of Spread

The food industry (malting, milling) and segments of the feed industry (swine feeds) will continue to rely on grain with little or no FHB for their manufacturing processes. Much discussion centred around ways of maintaining this supply. Until disease resistant varieties are available, spread of *F. graminearum* must be minimized. Producer and industry education was seen to be the key – in areas where *F. graminearum* is presently rare, growers should use seed free, or essentially free, of this pathogen as well as a seed treatment effective against *F. graminearum*. Efficacy of various seed treatments should be more widely known. It was pointed out that the Seeds Act does not require labeling for *Fusarium*. There is, however, a requirement under the Seeds Act to label grain as "not tested for smut" and there was discussion as to whether a similar requirement be added for *F. graminearum*. It was generally felt that changing legislation is a long term process and is not a viable option.

NEEDS/PRIORITIES

The spread of *F. graminearum* must continue to be monitored. Producer education, through expanded extension programs, must continue to emphasize recommended management practices.

BIOTECHNOLOGY

Facilitator: Therese Ouellet

The current biotechnology efforts to improve FHB resistance can be grouped in two areas: mapping/cloning and transformation. Gene cloning/search through mapping is going on in wheat and maize to identify QTLs for FHB resistance and potential resistance (R) genes. Eleven markers corresponding to 3 QTLs and 3 potential R genes have been identified by the CRC, Winnipeg, in resistant wheat and are being developed for marker-assisted selection. Cloning of these potential R genes will be attempted. Markers for QTLs associated with resistance have also been identified in wheat by American groups and in maize at the University of Guelph. ECORC, Ottawa, with collaboration from the CRC have initiated a large scale effort using a EST approach and DNA chip technology to identify genes induced during plant/Fusarium interaction in wheat, maize and Fusarium. The goals are to increase our understanding of the plant/*Fusarium* interactions, identify genes associated with the resistance in different resistant cultivars as well as potential genes (not associated with DON synthesis) involved in pathogenicity in *Fusarium*.

A transgenic approach is also being used for wheat and maize to modify/create pathways aiming at improving resistance in the crops. Examples include the use of anti-fungal proteins, general host resistance genes (ex. oxalate oxidase, extensin, hydroflavone reductase, phytoalexins, etc), a trypsin inhibitor targeting fungal protease(s), DON-detoxifying enzymes of microbial origin, modification of the peptidyl transferase in the plant (the cellular target for DON).

Additional efforts are needed to take full advantage of the opportunities in biotechnology, and to complement and bridge the gap between current efforts in biotechnology, pathology and breeding. These include:

-Develop breeding material (large double haploid populations, near isogenic lines, etc) from distinct sources of resistance and specifically geared towards molecular needs. Sumai3 is used almost exclusively as source of resistance in wheat programs worldwide.

-Increase the characterization of the phenotypes in different sources of resistance to improve our understanding of genetic, physiological, biochemical and cellular aspects of the resistance in different sources. The association of the known resistance phenotypes (types 1 to 5) to specific mechanisms would allow the development of improved screening procedures for the selection of resistance. The breeding and mapping efforts have shown that Fusarium resistances are multigenic. Can they be described as the sum of specific and measurable components?

-Additional work on plant/*Fusarium* interaction at the structural, biochemical, molecular, etc, levels. This would increase our "set of tools" to describe more precisely the resistance types.

-Additional research on the pathogen, *Fusarium graminearum*, including public effort to sequence its whole genome and comparisons with other *Fusarium* pathogens of cereals.

2) STRATEGIES FOR SOLVING FHB PROBLEMS

Chair: Harvey Voldeng

Summary of General Discussion and Resolutions

WHEAT

Moderator- Jeannie Gilbert
Minutes- Tim Paulitz

The session gave the opportunity for researchers working on wheat to identify research needs and areas for potential collaboration.
The Wheat group suggested that the following areas need more research:

- Finding resistance mechanisms
- disease forecasting
- sources of resistance other than Sumai 3, and its Ning derivatives.
- spread of disease, epidemiology
- studies on variation among isolates of the fungal pathogen
- improving methodology in disease screening nurseries
- biocontrol and antagonistic organisms
- most efficient methods for marker assisted selection
- fungicide X cultivar interactions
- increased capacity for screening cultivars

These suggestions were grouped into three broad categories

1. Applied work (in combination with mechanisms of host pathogen interactions).
2. Germplasm development
3. Agronomy
4. Collaboration
5. Goals

1. Applied Research
fungicide X cultivar interactions
screening methodology in nurseries
forecasting
marker assisted selection
species variability (pathogen

Nursery and screening protocols
Screening protocols have application over several areas, but we have difficulty in controlling experimental error and obtaining reproducible results. It is possible that we have to look at infection behavior and resistance mechanisms- host pathogen interaction at the basic level.

Forecasting
Manitoba Agriculture, University of Guelph ON, and AAFC are working on developing a better understanding of the weather conditions that promote FHB development. In the long term to be able to predict problems with FHB and help producers manage the disease.

Mycotoxin Analysis
A good method of DON analysis is needed by all commodity groups. Dr. Dorrell, Director General for western region has agreed to provide 0.5 PY to ECORC to help run the mycotoxin lab so that DON analyses can be done at reasonable cost.

2. Germplasm Development
- basic research host-pathogen interactions
- mechanisms of resistance
- rating inconsistency after inoculation
FHB resistance from wide crosses and alien species

3. Agronomy
- timing of seeding
- rotation
- tillage effects
Some collaborative work has been started at between Brandon MB, and Lacombe AB.

4. Collaboration
Biotech- more collaboration needed in this area, interaction, incentive to collaborate- It was pointed out that AAFC's Plant Genomic Initiative has a strong FHB component and could be another way of linking people.

As a means of facilitating collaborative links between researchers Randy Clear will put a page in the proceedings - a survey of attendees of the members. Those who wish to make their affiliation and areas of expertise known can respond to Randy and have their names included on the FHB web site.

5. Goals
-2 years from now, should have a funding program and collaborative research.
- some members of the group pointed out that we have defined objectives, but need funding

Funding:

Suggestions from the floor on the subject of research funding included:

- Under the auspices of the CWFHB a letter should be sent to all the funding agencies.
- There should also be a lobby group from the food industry (which has more lobby power than agriculture), informing the public of the potential safety hazards of FHB and mycotoxin contamination, as well as funding agencies. The importance of this was stressed several times during the discussion and the need to expand research on FHB.

- Information was offered about how in eastern Canada, the FHB problem was growing, but experts were being lost at the same time.

- There is a need to maintain expertise and training. Scientists need to have a voice and there needs to be effective networking with producers, processors, scientists, and marketers.

- It was suggested that we could take the problem to the public? Food safety and health. Like Greenpeace and Sierra Club.

U.S. experience.
- Model of the U.S. Scab Initiative was outlined: had a two stage process- 1. a steering committee met and set priorities among groups. 2. A small (2 members) executive committee took it further. After the steering committee was established but before lobbying, occurred, members of the committee went back to their respective state politicians to inform them of the issues around FHB and to ensure their support. Then when they went to the federal level to request funding for the Scab Initiative, the committee already knew they had the support of state politicians.

- The Wheat group decided that a resolution should be drafted stating that the organizing committee of the CWFHB should bring the issues of fusarium research and funding forward to politicians and to the agriculture industry. (editors note: please see the Meeting Conclusion on page 107 for the resolution)

BARLEY

Facilitator - Brian Rossnagel
Recorder - Jim Anderson

The barley breakout session was attended by more than 30 persons.

The group agreed to divide the time available into two sections with approximately one-third of the time allotted to "agronomic" issues and two-thirds of the time allotted to discussing "breeding/genetics/biotech/pathology" issues.

General points:

FHB in barley is very different than FHB in wheat, both in terms of the effects of the disease per se, but also in the concerns of the marketplace, especially for brewing. It was agreed that the brewing industries current standards of zero vomitoxin are not realistic.

The group agreed unanimously that unfettered collaboration was a key to any national strategy for R&D to combat FHB in barley with full sharing of responsibilities, costs, germplasm, technology and credit, unencumbered by parochial or institutional egotism, intellectual property and like issues.

The group supported the concept of learning from our USA colleagues who have been at war with FHB for considerably longer than we have. This learning should not only be at the scientific level, but in terms of the administration and functioning of a large inter-institutional collaborative effort such as that being proposed for a Canadian FHB plan.

It was suggested that FHB was the "common cold of cereals", which may be a good analogy to keep in mind when trying to design R&D to combat this disease. It was noted that the first resistant varieties will likely be compromised for other performance and quality traits.

A key point was that FHB agronomic issues and extension/education are essential components in "buying time" to enable breeding/genetic efforts to be able to take effect in combating FHB.

Agronomic issues:

Seed movement
Primary discussion points revolved around the need to attempt to prevent or at least minimize and slow FHB's western spread beyond Manitoba and SE Sask. The group stopped short of suggesting a quarantine approach and took the softer approach that seed purchasers and barley growers outside currently affected areas should be strongly advised to consider the possible spread of FHB in seed from infected regions and to consider growing varieties with the best available tolerance to FHB. Concern was also expressed

regarding the movement of FHB contaminated feed supplies from MB to AB.

Seed treatment

While the value of chemical seed treatment was considered important, concerns were expressed about the absolute effectiveness of currently available chemicals and the fact that even if effective treatments are often not properly applied resulting in potential seed transmission of FHB. More date is needed on efficacy when used specifically on barley.

Seed analysis

Concern was expressed with current seed testing labs' ability to accurately evaluate seedborne FHB and furthermore with the current lack of capacity in Canadian seed testing labs to handle the potential numbers of samples which might require analysis. Standard procedures need to be developed for seed lot FHB analysis.

Debris management

Consideration should be given to the use of debris burying by ploughing as part of an integrated approach to the management of the FHB problem. Despite soil conservation concerns, ploughing deserves consideration along with other less than desirable management practices such as the application of foliar fungicides. The need for complete burial of residue and good crop rotation was noted.

Foliar fungicide treatment

A primary concern is that little barley-specific information is available regarding the effectiveness of various fungicides for FHB control and extrapolation from wheat studies may have limitations. Recommended fungicides will only assist in alleviating the problem if they are applied appropriately and better information is needed by producers re timing of application etc. Concern was expressed that the recommendation to use fungicides to control FHB might lead to the general, undesirable, prophylactic use of fungicides for overall disease control, leading to concerns about the image of our produce in the marketplace and a general move away from efforts to enhance general disease resistance in barley.

Other hosts

The movement of early maturing maize into western Canada could be a possible source of additional infection and spread of FHB. In addition it was pointed out that, even if new maize varieties are resistant to FHB, they still may act as carriers and spreaders of FHB.

Extension/Producer Education

A key point raised in the discussion was the need to educate producers and public and private extension agents currently in the non-affected portions of western Canada about the serious potential risk from FHB. Reference was made to the NIMBY (not in my backyard) syndrome being a current problem and that people in these as yet non-affected areas need to be brought up to speed with regard to the threat of FHB, methods to prevent its introduction, early recognition of the disease and the like.

Crop models for fungicide application
Some discussion took place regarding the potential value of a form of an "early warning system" for FHB which, if available, would be a very useful management tool for farmers. The analogy was drawn to the potato late blight warning system currently in place for Manitoba.

Breeding/genetics/biotech/pathology

Phenotyping disease
Agreed that the key difference for barley was the extreme difficulty in accurately and consistently phenotyping FHB in field nurseries. It was generally agreed that interference from other diseases, specifically spot blotch, the generally lower level of infection in barley and the fact that barley grain does not exhibit tombstone-like symptoms make it more difficult to work with than wheat. Consequently several persons expressed the opinion that DON measurement was specifically more important for FHB resistance R&D in barley. The fact that the most critical effect of FHB in barley, whether for feed or brewing, is contamination with DON, rather than effects on grain yield or physical quality, also leads to a greater focus on DON measurement rather than field evaluation for FHB per se. These issues affect identification of resistant germplasm, the actual breeding for resistance and the associated screening as well as evaluation of existing varieties and quality control testing of producer samples. It was also pointed out that accurate phenotyping is absolutely critical if any success is to be achieved in the development of molecular markers to assist in breeding and if any type of genetic engineering solutions are to be considered.

Evaluation
There is a great need to properly evaluate existing varieties for FHB reaction and to add that information to provincial variety info pamphlets. This is important so that farmers in currently affected areas can make the best variety choices to minimize their risk, and perhaps even more important for producers in currently non-affected areas, to help them minimize the risk of FHB becoming a problem.

DON detection
Given the critical nature of DON as the key problem caused by FHB in barley there is great need to develop faster and much less expensive DON detection methods. Not only is the current cost per sample a major limitation, but lab capacity is also lacking. It was noted that different methodology could be used for screening/breeding versus that needed for a quality control type of situation. For breeding and screening work absolute accuracy can be compromised for efficiency since materials will be screened several times before decisions are made and breeding work can tolerate higher levels of type II errors than can quality control. There was some discussion of possible alternative methods to measure DON - e.g., since pigs can detect these compounds based on some

level of volatiles, is there anything that could be learned and developed from that in terms of a screening method?

Disease nurseries

Despite field symptoms being less valuable than for wheat, field screening nurseries are critical for barley and current levels of funding are woefully inadequate for screening, evaluation and breeding purposes. There is also a need to optimize field and indoor FHB screening methodology. Nurseries in China appear to be effective for our US counterparts and could be efficient as they are off-season and could be relatively inexpensive to operate. Furthermore, collaboration with Chinese groups might lead to increased access to resistant germplasm from those areas. Due to confounding of current Manitoba nurseries with spot blotch there was brief discussion of alternative sites. Possibilities include - areas of Quebec (although spot blotch and other disease may confound FHB testing), the southern Alberta irrigation area (FHB already endemic there) or possibly isolated areas where FHB and other confounding barley diseases are generally not a problem but where FHB could be developed and maintained in isolation. CIMMYT nurseries in Mexico would be a possibility, but current space limitations for barley may make that difficult.

Resistance sources

Bad news - in 6-rows and despite much effort in US not a lot of progress to date. Good news - in 2-rows it appears that several current western Canadian varieties have reasonably good FHB tolerance. Good evidence that both morphology and physiology are involved meaning there are several avenues of R&D to be followed. Hulless barley may offer lower DON levels since at least a portion of the DON will be left in the field, however, this is only avoidance and the hulless trait must be combined with resistance to be effective in the long term. Unfortunately most resistant sources from the Far East have very poor agronomics. May be useful resistance in materials from CIMMYT and South America, especially Uruguay. ***More screening of introduction material needs to done.*** Molecular and morphological marker development is possible and would be desirable. Need to make best use of doubled haploidy and SSD to develop marker populations. Should consider a planned/shared approach to crosses to be evaluated since resources will limit ability to screen large numbers. Need to intercross local tolerant 2-rows to determine if additive gene action exists to improve resistance. Need to work on both 2-rows and 6-rows. Several breeding programs currently have populations from crosses with "resistant" Chinese introductions at a stage where screening needs are imminent.

Gene pools

Time limited discussion in this area, but it was agreed that potential sources of resistance in both the secondary and tertiary barley gene pools should be evaluated. Concern was expressed that pending retirements in AAFC might lead to no one left in Canada with a mandate for such activity, especially with regard to barley. Once again limited screening resources negatively affect this effort.

Genetic Engineering

Genetic engineering was looked on as a potential long term solution , both because of difficulties in transforming barley and also because of current acceptance of GMO's, but it is noted that at least two programs in Canada are actively working towards transforming barley with ECORC FHB resistance genes.

Molecular marker development

It was unanimously agreed that unfettered collaboration among Canadian barley workers will be critical to success in this area. All aspects including workload, obtaining support, costs, germplasm, markers and credit must be shared. A strategy for sharing costs/workload/credit should be developed with those wishing to take part agreeing to this concept. Doubled haploid and RIL populations should be developed and checked against US markers. We should intercross current good 2 rows to determine if it might be possible to pyramid quantitative resistance. Use 2x haploidy wherever possible. Strategic populations should be co-developed keeping in mind NABGMAP project interactions and direct interaction with USA colleagues.

In addition to looking at molecular approaches to assist in breeding for FHB resistance it was suggested that molecular approaches could be considered to better understand the FHB pathogen, but the group was reminded that much of this work is already underway in the US and should not be duplicated.

CORN AND OATS

Facilitator - Art Schaafsma
Secretary - Jennifer Mitchell Fetch

This session was attended by 11 participants interested in the problems caused in oats and corn by Fusarium infections.

General Discussion

Feeding infected grain to livestock

In Ontario in 1999-2000 infected corn is being fed to hogs. Some samples contain up to 6 ppm of DON. Rations are formulated with about 14 kg per tonne of an additive that appears to render the feed acceptable to hogs. No claims are made for the additive, but it is being used. It may not need to be registered as no claims are made. No further details were available.

DON analyses

Samples from research projects can be analyzed for DON at the Eastern Cereal and Oilseed Research Centre (ECORC) for $5.00 a sample. Further details can be obtained from Dr. Marc Savard (613-759-1683).

Epidemiology

There is a need for histopathology studies to determine the manner of infection of corn

(and other cereals) by the fungus. This is not clearly understood, especially the relative importance of different times and tissues of infection.

Residual Management

There was debate about the importance of residual management method of controlling the fungus. In general the residual does not seem to contribute to increased epidemics. An exception might be when wheat follows corn, which may result in more point inoculation and infection. Participants noted that the inoculum source is everywhere. Infection studies have been done on some fields for three years and there appeared to be no increase in infections or spores. So called 'hot spots' are related to environmental conditions in that area. There was some discussion as to what has altered the 'window' that allows development of epidemics:

-varieties have changes?
-the fungal population has changed?
-global climate shift?

Mycotoxin studies

There was discussion of the role of mycotoxins in infected plants. The fungus can apparently survive its own toxin, the plant cells cannot. But the hyphal tip is where the toxin accumulates, and someone noted that that area is essentially 'dead'. The possibility of turning the toxin on the fungus was discussed.

Molecular markers

The need to link molecular markers to genes for resistance was considered important in order to increase the efficiency and effectiveness of breeding efforts. This would be aided by the development and use of doubled haploid populations. [It would also be aided by improved phenotypic classification of resistance and susceptibility - editor].

Oats

There is a reduction of the DON level in oats after steam processing. Is the DON washed off or just inactivated?
What is the extent of the FHB problem in hulless oats?
Does *Fusarium moniliforme* infect small grain cereals?

Needs Identified

1. Genetic resistance in our varieties
2. Studies of the pathogen and epidemiology.
3. Role of crop residue in disease development (some debate on this).
4. Models to forecast disease outbreaks.
5. Ways to mitigate the effects of mycotoxins in feed and food.
6. Biological control: There are possibilities but it may be better to isolate the bacterial genes of interest and put them in the crops by molecular transformation.
7. Detoxification of the mycotoxins.

8. Molecular markers for resistance
9. Extent of FHB infection of oats in North America

CONSUMER ISSUES

Facilitator - Daryl Embury

The need to educate consumers was emphasized, not only about the possible effects of Fusarium mycotoxins, but more importantly, the safeguards in place to ensure that harmful levels do not enter the food system.

There is also a need for international agreements on allowable levels of DON and other mycotoxins in commodities and by-products.

Meeting Conclusion

At the end of the meeting, a single final resolution was proposed to the Main Group. It was moved by Julie Gold and seconded by Jeannie Gilbert. The motion passed unanimously.

Motion:

Be it moved that a steering committee be struck for the purpose of bringing issues of fusarium head blight research (including all cereals and other commodities) to legislative and funding bodies.

The committee would, in particular, provide information and emphasize the seriousness of the problem and the need for ongoing funding for comprehensive, collaborative research and consumer education.

The committee should be comprised of research and extension scientists, producers, processors, consumers and marketers, who will appoint an executive committee responsible for taking action.

Poster Abstracts

Effect of *Microsphaeropsis* sp. strain P130A on the field production of perithecia of *Gibberella zeae* on wheat residues

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Gibberella zeae (anamorph: *Fusarium graminearum*), the causal agent of fusarium head blight, can overwinter as perithecia on wheat residues which serve as the main source of inoculum the following year. *Microsphaeropsis* sp. strain P130A, is an antagonist of *Venturia inaequalis* that attacks the pseudothecia during the winter, and consequently reduces the initial inoculum. This antagonist was tested as a post-harvest application for its ability to reduce the number of perithecia of G. *zeae* on wheat residue. The biocontrol agent was applied on the residue with three application timings: in the fall, in the spring and both in the fall and spring. The number of immature and mature perithecia was counted on both straw and spikelet residues. When applied on the residue, the biocontrol agent reduced the number of perithecia, with varying efficacy depending on the sampling date. In July, at anthesis of the wheat plant, the antagonist reduced the number of perithecia on both types of residue. The results of this first experiment are encouraging, however, much more work needs to be done for optimising the efficacy of the biocontrol agent, including dose, formulation and timing of application.

Mycotoxins in eastern Canada's barley and oats

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In Eastern Canada *Fusarium* species infect barley more frequently than wheat, yet information on mycotoxin contamination in barley is lacking. Such information is essential to establish a bench mark for mycotoxin contamination and to determine the need for control of fusarium head blight in barley. Therefore, data were retrieved from the Mycotoxin Databank of the Canadian Food Inspection Agency, Feed Section to study mycotoxin contamination in Eastern Canada's barley and oats. Of the 116 barley samples studied, 84 (72%) were contaminated with vomitoxin. Some samples contained up to 8-9 mg/kg of vomitoxin. Vomitoxin contamination was particularly severe in recent years (1996, 1997, and 1998). Vomitoxin contamination was less frequent and less severe in oats in comparison with barley. Only 34 of the 72 oat samples (47%) contained vomitoxin. Forty-nine percent of the barley samples (18/37) and 17% of the oat samples (4/24) contained nivalenol. Seventeen percent of the barley samples (17/100) and 1% of the oat samples (1/70) contained zearalenone. Six percent of the barley samples (6/94)

and 3% of the oat samples (2/69) contained ochratoxin A. Three barley samples were contaminated with 3-acetyldeoxynivalenol, and two barley samples with 15-acetoxyscirpenol. One barley and one oat sample each was contaminated with T-2. HT-2, diacetoxyscirpenol, fusarenon X, 15-acetoxyscirpenol, and neosolaniol were not detected in these barley and oat samples. The results suggest that breeding for resistance to vomitoxin accumulation is warranted for barley in Eastern Canada.

Seedling susceptibility of roots of crop species to a foliar isolate of *Fusarium graminearum* of wheat

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Fusarium head blight, caused primarily by *Fusarium graminearum*, is an important disease of wheat in western Canada. Previously confined to Manitoba, the disease has occurred in more westerly regions in recent years. To understand more about possible mechanisms of spread, a study was conducted to determine whether *F. graminearum* isolated from wheat heads can infect roots of wheat, barley, oat, rye, triticale, canaryseed, *Brassica napus*, *B. rapa*, *B. juncea*, bean, pea, lentil and chickpea. One spikelet of Roblin wheat infected with a single *F. graminearum* isolate, was placed adjacent to seeds of each crop at seeding. Emergence was counted and disease symptoms on roots or seed infection scored 4 weeks later. The effect of temperature on infection was also tested on barley cv. Brier. Reduction in emergence was significant in all crops except for *Brassica* species and pea. Inoculation resulted in significantly more root infections in most crops. At the lowest temperature of 10/5°C (day/night), no infection occurred on barley and emergence was significantly delayed. However, as the temperature increased from 10-30°C, time to emergence became progressively shorter and infections increased in a quadratic manner. This study shows that *F. graminearum* is favored by temperatures between 20-30°C and that this pathogen can infect several crop species. These results have significant implications on the epidemiology of this disease and on crop rotation as a strategy for disease management.

A progress in resistance to FHB in Quebec wheat cultivars

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Quebec was a wheat producing province until the wheat midge (Sitodiplosis mosellana) led to crop failures, circa 1840. Novel cultivars with high yield renewed interest in wheat culture after 1970; unfortunately, by 1980, FHB had become a major problem in feed wheat. A bread wheat breeding project was initiated at AAC Ste-Foy in 1982, with collaboration from the MAPAQ pathologist. A first resistant cultivar was released in 1994 and four more afterwards, by the time the program was terminated in 1997. The five cultivars were based on a rather diverse background, yet the resistance was obtained strictly from Quebec and Manitoba germplasm sources. The resistance level achieved was moderate but useful, similar to that of Neepawa and Katepwa. One cultivar (AC Pollet) was shown to have a narrower sensitivity window at flowering. Evidence of the effectiveness of the resistance of Quebec cultivars in epidemics will be shown. It is now demonstrated that some progress against FHB can be obtained without the use of foreign germplasm, and also that some transgressive segregants are more resistant than their parents. It is hypothesized that some of the FHB resistance might relate to resistance to the midge.

Resistance to FHB associated with the length of rachis internodes

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Morphological traits have been recently related to FHB. To investigate the role of compactness, nine intergeneric and three standard bread wheat lines were inoculated with conidial suspensions of *Fusarium graminearum*. A negative correlation (r=-0.74; p<0.0006) was found between the length of the rachis internodes and the spread of FHB. Within the series of intergeneric *T. durum* Agropyron distichum* (*Elytrigia disticha*) lines, 3 lines resisted FHB better than Nyu Bay, the resistant check. This study showed that the spike morphology could be a more valuable morphological character to study, as it involves a good biodiversity for many traits, many of which might be somehow linked to disease reaction, including FHB. Pleiotropy, linkage and additive effects with detoxification-related alleles cannot be ruled out.

Fusarium spp. isolated from heads and roots of wheat in Saskatchewan and their ability to cause Fusarium head blight

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A study to identify root pathogens, determine their distribution in different soil types, and compare isolates of *Fusarium* spp. from roots and heads of wheat, was conducted in Saskatchewan in 1998 and 1999. Most of the *Fusarium* spp. isolated from infected heads were also found in the subcrown internodes of plants collected in the same fields, although at different relative frequencies. Overall, *F. poae, F. graminearum, and F. sporotrichioides* were found at higher levels in subcrown internodes than heads, whereas *F. equiseti* and *F. culmorum* were more common in heads than subcrown internodes. This suggests that inoculum in debris from plant parts at or below soil level might be a source of infection for heads, and/or that infected heads/kernels might be contributing to root infections. Controlled environment tests to determine the pathogenicity of *Fusarium* spp. to heads showed that *F. graminearum* and *F. culmorum* were the most pathogenic. For each of the species tested, isolates from heads and subcrown internodes were equally pathogenic to wheat heads. Most of the *F. graminearum* isolates from subcrown internodes produced perithecia. The significance of these observations in the control of FHB in Saskatchewan is discussed.

Fusarium head blight in wheat and barley in Saskatchewan in 1998 and 1999

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A province-wide survey of barley and common and durum wheat was conducted in Saskatchewan in 1998 and 1999 to determine the incidence and severity of fusarium head blight (FHB). This disease was found in most crop districts surveyed. Overall, the percentage of infected fields in 1998 and 1999 was 60% and 65% for barley, and 55% and 50% for wheat (common and durum). Percent fields infected was lowest in Zone I (Brown soil) in the southwest and highest in Zone III (Black/Grey soil) in the east and north. In general, disease severity was low. The average FHB index was also lower in 1999 than in 1998 for all crops (1.4-2.8% in 1998, 1.0% in 1999). For both years, it was lowest in Zone I (0.1-0.7%) and highest in Zone III (1.0-3.4%). *Fusarium poae* was the species most frequently isolated from infected heads, followed by *F. sporotrichioides*, *F. graminearum*, and *F. avenaceum*. Higher levels of *F. avenaceum*, and lower levels of *F. graminearum*, in 1999 than in 1998 was attributed to cool weather prevalent in most of the province during the 1999 growing season. *Fusarium culmorum* and *F. equiseti* were found at lower frequencies than the above. In both years, *F. avenaceum* and *F. culmorum*

were more common in wheat, whereas *F. poae* was more common in barley.

A novel method to identify *Fusarium* spp. that attack roots of wheat and barley seedlings

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Roots of wheat and barley seedlings grown from fusarium head blight (FHB)-contaminated seed, were examined for *Fusarium* species using a novel method. Seed of three varieties each of FHB-contaminated wheat and barley were grown in a split plot design with 4 replications. Treatments included two seed dressings and a control. At GS 15-25 (end of tillering) whole roots were dug up, thoroughly washed in running water and frozen for later examination for infection by *Fusarium* spp. Roots were surface-sterilized in 0.1% NaOCl for 1 m and, without rinsing, allowed to dry for two hours on sterile filter paper in a laminar flow hood. Roots and filter paper were then placed on PDA agar and left overnight. A film of cooled Komada's medium was poured over the roots and the plates incubated under continuous cool white fluorescent light for 5 to 7 days. Colonies formed were identified to species using dissecting and compound microscopes. Five roots per plot were examined for *Fusarium* species. There were no significant treatment or variety differences. Nine species of *Fusarium* were isolated of which the predominant ones from wheat were *F. equiseti* (43.7%), *F. graminearum* (17.9%), and *F. sambucinum* (13.3%).

Biological inactivation of trichothecenes - Development of an in vitro model with pig's intestine

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The contamination of grain with *Fusarium* toxins represents an unavoidable and worldwide problem in the feed industries. Therefore the aim of a several years' study was the development of a feed additive that detoxifies diet containing trichothecenes (in particular deoxynivalenol and its acetylated metabolites) directly in the upper part of the animal's intestine.
The gained product *Biomin® BBSH 797* bases on viable microbes that were isolated from rumen contents. Their bacterial epoxidases are able to biotransform trichothecenes by selective cleavage of the epoxide group, resulting in harmless metabolites.
For the determination of product-stability (i. e. preservation of biotransformation-activity) in a practically relevant habitat as well as for the estimation of an optimum product concentration in subsequent feeding trials an *in vitro* model with pig's intestine was developed and tested for its usability.

The deepoxylation-activity of the strain could be verified under the conditions of the gut environment. The existing gut microflora as well as the physical gut conditions turned out to be suitable for the biotransformation of trichothecenes.

Survival of *Fusarium graminearum* on fusarium damaged kernels

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The role and potential contribution of seed borne *Fusarium graminearum* to fusarium head blight (FHB) outbreaks is not well understood. To investigate this aspect of epidemiology, fusarium damaged kernels (FDK) were separated from healthy seeds in FHB contaminated grain samples of two spring wheat cultivars, Roblin and AC Domain, and placed into small nylon mesh bags. Under field conditions, bags were left exposed on the soil surface or buried at either 5 or 10 cm at Glenlea Research Farm, MB, in a 4 replicate randomized complete block design (1 bag, 50 FDK per replicate). Bags were harvested at 6 months and 1 year and will continue at 6 month intervals for a second year to determine the survival of *F. graminearum*. The same experimental design was used for controlled conditions, but each bag contained 25 FDK. Bags were left on the surface or buried at 5 cm in sterile or non-sterile soil and exposed to a constant temperature of 20EC, 5EC, or -10EC. Four bags per treatment were harvested at 6 months and 1 year and will continue for a second year as for the field study. Survival of *F. graminearum* ranged from 87.8-100 % over all treatments in both the field and under controlled conditions after 1 year incubation. Greatest loss of viability occurred under controlled conditions at 20EC in non-sterile soil.

Rapid detection of FDK using digital image analysis and neural networks

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A digital image analysis system has been developed to detect and quantify FDK (Fusarium Damaged Kernels) in wheat. The system provides for objective and repeatable analysis of large grain samples (500 to 2,000 seeds). Sampling is rapid (less than 2 minutes) and it is accurate. The system is based on modified non-video scanning device technology employing high resolution 24-bit colour imaging. It is relatively inexpensive, having familiar PC-based hardware and software. The system incorporates artificial intelligence (AI) modules, with a combination of neural networks and expert systems. It can be regarded as an electronic supplement (tool) to augment the present visual evaluation system and can be incorporated as a component of an integrated food safety monitoring system. It generates exportable digital sample-linked reports (files, data, images) without transcription errors. It does not involve consumable supplies, is non-destructive to samples, and does not require time-consuming sample preparation. Providing objective quantification of Fusarium Head Blight in seed lots is seen as a benefit to breeders and pathologists. It can facilitate product differentiation and segregation in the market place.

Evolutionary potential of *Fusarium graminearum* : a risk for the future ?

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Fusarium head blight, caused by *Fusarium graminearum*, was not even mentioned in most plant pathology textbooks in the 50's. Nowadays it is considered as one of the most important problems of wheat in North America. We are attempting here to define and understand the possible threat associated with the evolutionary potential of *F. graminearum*. The evolutionary tendency of some strains is enhanced by a selective pressure restricting the adaptive value of other strains and a new opportunity or potential niche allowing expression of existing adaptive genes. An increased presence and damage by *F. graminearum* could be related to changes in: 1) competitive ability, 2) adaptation to abiotic environmental stress, 3) toxin production, 4) sexual recombination and 5) timing of ascospores ripening. Evolution rate may vary, but modern agriculture does cause selective pressure and also opens new niches, perhaps resulting in increased fitness of certain alleles or pathogenic strains which may tend to become dominant in the environment. This discussion on biodiversity within *F. graminearum* indicates a need for research on the impact of cultural practices and plant breeding on the genetical make-up of this pathogen population. This also shows that more information is required on the possibility that some resistance alleles might cause a selective pressure favoring more aggressive and toxigenic *F. graminearum* strains.

Relationship between Fusarium head blight and common root of wheat and barley in Quebec

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In Quebec, *Fusarium graminearum* is one of the most common causal agents of Fusarium head blight (FHB). It is also, together with *Bipolaris sorokiniana*, a causal agent of common root rot. *Fusarium* spp. and *B. sorokiniana* were isolated from wheat and barley seeds in 1997 and from roots in 1998. *F. graminearum* accounted for 33% and 52% of *Fusarium* isolates from seeds and roots, respectively. Most of the *F. graminearum* isolates were more pathogenic on wheat seedlings in test tubes than *B. sorokiniana* isolates. The *F. graminearum* isolates from roots and those from diseased kernels shared a similar capacity to cause FHB on wheat in a greenhouse trial. All these isolates formed perithecia *in vitro* on dead seedlings, indicating that they are *F. graminearum* and not *F. pseudograminearum* (formerly recognized as *F. graminearum* group 1). In strategies to control FHB, it seems essential to also consider the common root rot caused by *F. graminearum* as an important part of the disease epidemiology. The role of seedborne

Fusarium in epidemiology also needs attention. We are dealing with a pathogen that can attack above and below ground level, and seeds could be a vehicle for its propagation.

Comparative ability of *Fusarium graminearum*, *Fusarium poae*, *Fusarium sporotrichioides*, and *Fusarium avenaceum* to cause head blight in barley

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Various *Fusarium* species can be isolated from barley seed with symptoms of fusarium head blight. To investigate their relative pathogenicity, the barley varieties AC Lacombe, AC Oxbow, Bedford, and Argyle were inoculated with conidial suspensions of *Fusarium graminearum*, *Fusarium poae*, *Fusarium sporotrichioides*, *Fusarium avenaceum*, and an equal mixture of all four species. The experiment was run under field conditions over two years. In year one, relative humidity and soil moisture were elevated during inoculation and all treatments reduced thousand kernel weight with *Fusarium graminearum* causing the greatest reduction and highest level of seed infection. In year two, the relative humidity and soil moisture were lower and none of the treatments reduced the thousand kernel weight relative to the control although all species colonized the heads. Even though plastic humidity 'tents' were used in both years, environmental variation between years may have accounted for the differences observed.

Evaluation of *Triticum turgidum* L. var. *dicoccoides* for resistance to Fusarium Head Blight and stem rust

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Fusarium Head Blight (FHB) is a severe disease problem on tetraploid durum wheat, and good sources of resistance have not been available. Sources of resistance to FHB are known in hexaploid wheat but attempts to transfer this resistance to durum have been so far unsuccessful. *Triticum turgidum* var. *dicoccoides* (TD) is a wild tetraploid wheat which shares the A&B genomes with durum and crosses readily with it. TD is known to be a source of resistance for several diseases but no information on its reaction to FHB. We tested 290 accessions of TD for reaction to FHB. Ten accessions were more resistant than the best available durum line. Six of these accessions showed stem rust resistance to one or more of 5 pathotypes, but none were resistant to the sixth pathotype, PgtTPM. At least 6 known Sr genes along with unidentified Sr resistance genes were postulated. It is apparent that TD will be useful in improving resistance in durum to both FHB and stem rust.

Distribution of *Fusarium* species in cereal samples from Saskatchewan tested at commercial laboratories in 1998 and 1999

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This information was compiled to supplement data for western Canada obtained by personnel in the Research Branch of Agriculture and Agri-Food Canada and the Canadian Grain Commission. The distribution of *Fusarium* species on cereal grain samples received at two commercial labs from September 1998 to November 1999 was tabulated according to crop district and crop kind (common wheat, durum and barley). The commonest species isolated was *F. avenaceum*, except on a small number of durum samples from S.E. Saskatchewan. Other species identified were *F. graminearum, F. sporotrichioides, F. poae, F. culmorum* and *F. equiseti*. Sample sizes were too small to demonstrate differences among crop kinds in frequency of different species. *Fusarium graminearum* was most commonly found on samples from S.E. Saskatchewan (Crop districts 1A and 1B). However, it was also found in three rural municipalities (R.M.s) in crop district 8A, two R.M.s in each of crop districts 5A, 5B, 6B and 9A and one R.M. in each of crop districts 2B and 3B-N. The records from crop districts 3B-N and 6B are all from irrigation areas.

Genes induced during early infection of maize ears by *Fusarium graminearum*

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Fusarium graminearum attacks a wide range of plant species including maize (ear and stalk rot), barley, and wheat (head blight). Favorable environmental conditions (conducive temperatures and high humidity) can result in Fusarium epidemics and millions of dollars lost in crop revenues. *F. graminearum* infection in the cereals reduces both grain yield and quality and also results in mycotoxin contamination. We have initiated a study of the molecular interactions between *F. graminearum* and maize during infection of the silk channel and ear in susceptible and resistant inbreds. Differential RNA display- RT-PCR has been used to identify genes, from *F. graminearum* and corn, that are elicited in the early stages of infection of maize silk by the fungus. Additionally, infection in resistant inbreds has been compared to that from highly susceptible inbreds using this technique. Unique cDNA fragments originating from either *F. graminearum* or *Zea mays* have been cloned and characterized. A summary of the findings will be presented.

Fungicide impact on wheat in Manitoba

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Introduction

Fusarium head blight (FHB) has had a major affect on wheat yields in Manitoba. FHB currently occurs throughout all crop regions and affects wheat crops wherever environmental conditions favour the disease. In 1999, wheat yield were reduced an average of four percent. Wetter than normal summers in the 1990's, have contributed to the incidence and severity of FHB. Currently registered cultivars have minimal resistance to combat this disease. In response to produces and industry concern over the lack of disease management tools available for controlling this disease, Manitoba Agriculture and Food supported the emergency registration of Folicur 432F (tebuconazole) for the 1999 production season.

Objective

To determine the impact of fungicides on wheat for the control of Fusarium head blight and leaf diseases including leaf rust, tan spot and Septoria leaf blotch.

Materials and Methods

Experimental Design:

A four replicate field scale (2.5 acres per plot) trial was conducted in the Central region in the vicinity of Carman.

Treatment:

Folicur 432F @ 118 ml/acre, Bravo 500 @ 0.8 L/acre, Dithane DG Rainshield NT @ 0.90 kg/acre and Tilt @ 0.2 L/acre. Fungicide applications of Folicur, Bravo and Dithane were at twenty-five percent anthesis stage. Tilt was applied at full flag leaf stage prior to head emergence.

Data Analysis

ANOVA to determine the significant difference between fungicide treatments. Mean averages followed by the same letter are not significantly different at P = 0.05, the 95% level of confidence.

Trials were evaluated for leaf disease infection, incidence of FDK parts per million of deoxynivalenol (DON) and yield. The FDK and DON analysis was done by SGS Canada Inc. in Winnipeg.

Yield results were based on weigh wagon measurements.

Results

Folicur treatment resulted in significantly less leaf disease, a combination of leaf rust and Septoria leaf blotch, than the treatments of Bravo, Dithane, Tilt or the untreated check as assessed on July 26th. The Tilt and Dithane treatments reduced leaf disease significantly more than Bravo. Bravo reduced leaf disease only in comparison to the untreated check.

Folicur, Dithane and Bravo significantly reduced the percent by weight of fusarium damaged kernels (FDK) and the parts per million of deoxynivalenol (DON) in the harvested grain as compared to the Tilt treatment or the untreated check. Folicur, Dithane and Tilt treatments resulted in significant yield increase over Bravo and the untreated check.

Conclusion

Folicur application resulted in a significant yield increase over the untreated check and Bravo treatment. Although the average yield increase was greater than that achieved by the Tilt and Dithane treatments, it was not significantly different at $P = 0.05$. The yield increase was found to be the result of a combination of the reduction in leaf disease and the reduction in the percentage of fusarium damaged kernels in the harvested grain. Folicur was the only fungicide that increased yield, reduced the percent of fusarium damaged kernels and reduced the amount of DON in the harvested grain. These findings for Folicur were supported by results of a small plot replicate cultivar trial that was conducted at Hamiota, Manitoba.

Sequential distribution of the mycotoxin deoxynivalenol in the different parts of wheat heads after inoculation with *Fusarium graminearum*

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One central spikelet of Roblin wheat spikes was inoculated with microspore suspensions of *Fusarium graminearum* and the entire spikes were harvested at 2 to 4 day intervals from 2 to 25 days after inoculation. These spikes were dissected and the amount of deoxynivalenol (DON) in each spikelet and each internode of the rachis was measured by ELISA. The first high concentrations of DON were found in the inoculated spikelets only 4 days after inoculation. DON concentrations in the spikelets below the inoculation point eventually reached 500-600 ppm while the corresponding internodes of the rachis contained 1000-1200 ppm. Relatively small amounts of DON were found above the inoculation points.

Optimizing fungicide application for Fusarium head blight (FHB) management in winter wheat in SW Ontario

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FOLICUR (tebuconazole) has been determined to be the best candidate fungicide for the management of FHB in winter wheat in Ontario. However it has its limitation. While it has acropetal translocative properties the timing of application require that it be considered a protectant. This paper looks at loading and distribution patterns for several nozzle and sprayer configurations to optimize spray coverage on wheat heads. The best configuration appear to be some form of two-way spraying such as twin jet nozzles or two nozzles (either flat fan or turbo jet) mounted forward and backward at each nozzle point. The interaction between FOLICUR and cultivar is also examined.

Maintaining Fusarium Head Blight resistance in spring wheat through successive breeding cycles

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Resistance in wheat to Fusarium Head Blight (FHB) is a character of highly complex inheritance. Introducing such a trait into commercial wheat and maintaining it through successive cycles of crossing to adapted but susceptible parents is a difficult task, requiring a disease testing procedure that is both intensive and reliable. For this purpose we combined extensive FHB screening in an irrigated field nursery inoculated with *Gibberella zeae* and intensive greenhouse testing of elite materials. We compared the FHB response in a set of lines representing progeny from first, second, third, and fourth breeding cycles of several different spring wheat crosses. Some first and second cycle progeny showed good FHB resistance but none combined that resistance with the agronomic traits needed for commercial release. A few third cycle and several fourth cycle derived lines combined those traits and some are candidates for release as commercial cultivars.

Fusarium Head Blight reaction of durum wheat lines conditioned by *Triticum dicoccoides* chromosome substitutions

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Fusarium head blight (FHB) is a serious disease problem on durum wheat. To date, the resistance to FHB available in hexaploid wheat sources has not successfully been transferred to durum -- a tetraploid wheat. *Triticum dicoccoides* is a wild tetraploid wheat that possesses many interesting traits. In the 1980's, USDA geneticist L.R. Joppa produced a set of lines derived from 'Langdon' durum, each with a different pair of chromosomes from *T. dicoccoides* substituted for the corresponding durum chromosomes. We tested these lines for FHB response by inoculation with *Fusarium graminearum* under controlled conditions. Two of the substitution lines were significantly less susceptible and two were significantly more susceptible than the durum parent, which itself showed a moderately susceptible FHB reaction. Since each line differs by an entire chromosome pair, the results suggest that FHB resistance genes compatible with the durum genome are present on at least four different chromosomes.

Varietal response in barley to Fusarium head blight

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Fusarium head blight (FHB) has become a serious and damaging disease of barley (*Hordeum vulgare*) in western Canada in the last 5 years. The principal causal agent is *Fusarium graminearum* but other *Fusarium* species, especially *F. poae*, may also be involved. As part of an integrated approach to FHB control, and to inform producers of cultivar performance to FHB, a group of 22 registered barley cultivars was evaluated in an inoculated nursery at Glenlea MB in 1998. Differences in response to FHB were found among cultivars, based on visual disease severity, *Fusarium* seed infestation and deoxynivalenol content. While a few cultivars had low or high scores for all parameters measured, correlations for the entire group were low, indicating the end-use of the harvested grain needs to be considered when determining which data to measure and analyze. Among the group of barleys tested, several two-rowed malting cultivars (AC Metcalfe, AC Oxbow, CDC Lager/CDC Kendall, CDC Stratus) and a six-rowed one (CDC Sisler) had superior over-all performance to FHB. The FHB responses of hulless barleys need to be interpreted with caution.

Preliminary evaluation of the potential spread of fusarium head blight, causal agent *Fusarium graminearum*, via infected feed grain

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Unfortunately, head blight caused by *Fusarium graminearum*, is no longer only a concern for Manitoba producers. In Alberta, surveys by the Canadian Grain Commission have found *F. graminearum* at trace levels in 10, 8, 14 and 70 wheat fields in 1995, 1996, 1997, and 1998, respectively. Recently, there have been reports of significant quantities of *F. graminearum* infected feed grain moving westward into Alberta to be fed to feedlot cattle. To assess the risk that this may be a method of introducing this pathogen into Alberta, a preliminary study was conducted looking at feed and manure samples from five Alberta feedlots where feed grain from outside Alberta was being used. Surface-sterilized intact and non-intact barley kernels from feed and manure samples were evaluated for the presence and viability of several plant pathogens, including *F. graminearum*. A variety of fungi were isolated from feed samples and spilled feed with the most common being *Alternaria* and *Penicillium* spp. Plant pathogenic fungi were also isolated and included *Pyrenophora teres, Cochliobolus sativus*, and a small number of *Fusarium* spp., but not *F. graminearum*. The amount of plant material screened from manure ranged from 63 to 122 g per kg of manure on a dry weight basis. For barley kernels screened from manure *Penicillium, Rhizopus/Mucor*, and *Aspergillus* spp. were the most common. The frequency of isolation of *Alternaria* spp. was greatly reduced, while no *P. teres* or *Fusarium* spp. were isolated from screened kernels, with *C. sativus* isolated from a single kernel. More research is needed to understand the potential for spread of plant pathogens via infected feed grain.

Seed-borne cereal disease survey, Alberta 1995-1997

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From 1995-1997, a cereal disease survey was conducted in the Peace River region and in an area from Edmonton south to Three Hills, Alberta. Approximately 30 heads were collected from each of 35, 32, and 94 barley and 28, 44, and 116 wheat fields in 1995, 1996, and 1997, respectively. Samples were collected nonselectively at several sites in each field at crop maturity. Seed infection was assessed for 100 seeds from each field using an agar plate procedure. Identification of fungi isolated from seed samples was based on colony and spore morphology. *Fusarium avenaceum* was the most common species of *Fusarium* isolated from both wheat and barley in 1995, 1996, and 1997, with average yearly seed infection levels of 1.1 and 3.3, 4.0 and 1.7, and 3.5 and 5.1%, respectively. No *F. graminearum* was isolated from tested seed in 1995, 1996, and 1997. *F. poae* was the second most common species recovered, although average wheat seed infection levels with *F. culmorum* were higher in 1997. The most common pathogens isolated from barley were *Pyrenophora teres* and *Stagonospora nodorum*, with average yearly infection levels that ranged from 7 to 20.5% and 10.5 to 19.5%, respectively. The most common pathogen isolated from sampled wheat kernels was *S. nodorum*, with average yearly infection levels ranging from 10.7 to 22%.

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